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SURGICAL TREATMENT OF FACIAL PARALYSIS*

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Paralysis of the seventh cranial, or facial nerve, caused by either disease or injury, is more common than paralysis of any other cranial or spinal nerve, and since facial paralysis produces asymmetry of the face, inability to close the eye, lacrimation, difficulty in mastication and deglutition, and loss of expression, it presents a serious problem to the otologist, the neurologist and the neurologic surgeon. The prognosis depends on the site and type of injury. Some lesions heal spontaneously, others need surgical intervention. As numerous operations have been proposed, I shall discuss the advantages and disadvantages of each and review a series of cases in which operation was performed at the Mayo Clinic.

ANATOMY OF THE FACIAL NERVE

The facial nerve, according to Stewart,[1] like the trigeminal is a mixed nerve, possessing a motor root, the facial nerve proper and a sensory root, the nervus intermedius of Wrisberg. These two roots meet at the geniculate ganglion. The motor root arises from a nucleus situated mainly in the lower part of the pons, some of whose cells (those for the orbicularis oculi) extend as high as the nucleus of the third nerve, and others (those for the orbicularis oris) as low as the hypoglossal nucleus. This root pursues a tortuous course. Within the substance of the pons it forms a loop which hooks round the nucleus of the sixth nerve, then leaving the ventral surface of the brain stem, it enters the internal auditory meatus, and passes along a winding bony canal in the temporal bone, the aqueduct of Fallopius or facial canal. The facial canal, as described by Ballance,[2] commences as a large foramen at the inferior part of the internal auditory meatus above the crista falciformis in the anterior and superior fossette. The canal passes at first horizontally

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1. Stewart, J. P.: Diagnosis of Nervous Diseases, Ed. 5, London, E. Arnold, 1920, 584 pp.

2. Ballance, C. A.: Surgery of the Temporal Bone, London, Macmillan and Company, 1919, pp. 612.

outward between the cochlea and the vestibule, then bends sharply backward anterior and superior to the fenestra ovalis, and runs horizontally a short distance, forming a visible projection on the inner wall of the tympanum. This ridge separates the inner wall of the tympanum proprium below from the inner wall of the attic above. Having passed close above the fenestra ovalis it turns inferiorly, and about 15 degrees laterally from the vertical, ending at the stylomastoid foramen on the inferior surface of the petrous bone. The nerve, as it emerges from the stylomastoid foramen, lies under cover of the anterior margin of the attachment of the posterior belly of the digastric muscle, the digastric groove here ending in the stylomastoid foramen. At the exit of the nerve the styloid process is mesial and a little anterior to it. In the upper part of this canal it traverses a swelling, the geniculate ganglion, which is joined by the sensory root or portio intermedia of Wrisberg, also by the great superficial petrosal nerve from Meckel's ganglion, and by the small superficial petrosal nerve from the otic ganglion. The otic ganglion, according to Stewart, is composed of nerve cells similar to those of the sympathetic ganglions; the geniculate ganglion is similar to a posterior root ganglion in structure, and is sensory in function. Inflammation of the geniculate ganglion is accompanied by herpes of the external auditory canal and adjacent part of the auricle, exactly analogous to herpes zoster.

Within the aqueduct the facial nerve gives off a branch to the stapedius, and lower down the chorda tympani leaves it to join the lingual nerve. It then emerges from the skull through the stylomastoid foramen, giving off a posterior auricular branch to the muscles of the pinna and to the occipital belly of the occipitofrontalis. The main trunk then divides into its terminal branches, supplying all the muscles of the face (except the levator palpebrae superioris) from the frontalis above to the platysma myoides below. It also supplies the stylohyoid and posterior belly of the digastric muscle.

The facial nerve contains two sets of afferent fibers: the taste fibers of the chorda tympani, from the anterior two thirds of the tongue, accompanying the facial nerve within the fallopian aqueduct, and the sensory fibers, which transmit deep pain sensations on pressure from all parts of the face. Both of these sets of afferent fibers lead to the geniculate ganglion and thence into the brain stem along the nervus intermedius. The facial nerve also contains efferent autonomic fibers to the sweat glands of the face and to the lachrymal gland in the orbit.

ETIOLOGY OF FACIAL PARALYSIS

A supranuclear paralysis may be produced by tumor, abscess, cerebral hemorrhage, encephalitis, meningitis or aneurysm. An isolated nuclear

paralysis is strongly suggestive of syphilis; occasionally it follows diphtheria. In epidemics of poliomyelitis the cranial nerves may be affected. Gibson,[3] quoting from a report of 338 cases observed at the Queensboro Hospital in New York during the 1916 epidemic, said that forty-six of this group showed involvement of the cranial nerves, twenty-six of which were of the facial nucleus and twelve of the sixth nucleus. Infranuclear lesions in the region of the pons are due to tumor, abscess, meningitis or hemorrhage, and are usually associated with involvement of other cranial nerves. Lesions within the facial canal are due to neuritis, otitic disease, tumor or trauma, either accidental, such as fracture of the temporal bone, or surgical, as from surgical interference in the treatment of mastoid disease. The latter type of trauma is relatively infrequent in the hands of a skilled operator. Lesions of the facial nerve below the facial canal are due to tumor, the most common type being parotic; to trauma, such as gunshot and stab wounds, and to incised wounds following drainage of infected glands in children, when the nerve is very superficial before being protected by the mastoid portion of the temporal bone.

The intracranial lesions producing facial paralysis will not be discussed, as they involve problems foreign to the subject of anastomosis of the seventh nerve, except when the patient, having recovered from the intracranial lesion, a residual facial paralysis, demands some form of anastomosis of the peripheral nerve.

The most common type of facial paralysis, Bell's palsy, is in all probability a simple neuritis, and is commonly supposed to be due to exposure to cold. Ney [4] believes that the lesion is situated in the vertical section of the facial canal, since the chorda tympani usually escapes involvement. In cases with involvement of the chorda tympani, it is rare, he believes, for the lesion to extend sufficiently high in the canal to produce paralysis of the stapedius muscle, and he uses this argument in recommending an anastomosis of the facial nerve in the canal when the nerve does not regenerate following Bell's palsy. The lesion itself is of a compressive nature, most likely due to inflammation and edema of the nerve, and heals spontaneously if the inflammation subsides without too much cicatrization and constriction.

Facial paralysis associated with acute or chronic otitis media is more common in children than in adults, but, according to Jones,[5] is rare as compared with the otitis media of childhood. When it does occur it is

3. Gibson, A.: Facial Paralysis, Surg., Gynec. & Obst. **33**:472-489 (Nov.) 1921.

4. Ney, K. W.: Facial Paralysis and Surgical Repair of Facial Nerve, Laryngoscope **32**:327-347 (May) 1922.

5. Jones, Barton: Quoted by Gibson.

brought about either by direct involvement of the nerve or by the pressure of exudate within the fallopian aqueduct, or by pressure from the tympanum. Caries of the wall may take place even in the acute stage. At necropsy the facial nerve has sometimes been found lying free in the tympanic cavity surrounded by pus, when there was no evidence of facial paralysis prior to death. From this he concludes that it is reasonable to infer that there must be pressure on the nerve within the narrow bony canal before facial paralysis is produced. Many cases of facial paralysis following acute mastoiditis have been reported in which there was subsequent improvement following a radical mastoid operation. Wallerstein's [6] report of a typical case illustrates this. The patient gave a history of acute otitis media which had ruptured spontaneously; five days after the onset facial paralysis began and became complete in two days. Following a radical mastoid operation there was immediate improvement, and in three days there was evidence of marked improvement. In this particular case there was no evidence of necrosis or dehiscence of the facial canal in spite of the facial paralysis, but Wallerstein believes that facial paralysis is more likely to occur when a dehiscence of the canal exists.

It is not uncommon for partial or complete facial paralysis to develop several days after a radical mastoid operation; this may be due to neuritis or edema of the facial nerve, or to a dehiscence of the facial canal which has permitted an extension of the infection into the facial canal; the latter usually subsides with recovery from the paralysis and subsidence of the mastoid disease. If the paralysis occurs immediately after the operation, it may be due to hemorrhage within the canal or to pressure or traction; only occasionally is it due to a complete severance of the nerve. Lillie [7] believes that the facial nerve is usually injured in the area on the promontory just anterior to the ampullar end of the horizontal canal in which the nerve comes around the eminentia pyramidalis; this makes it quite impossible to anastomose the nerve in the canal in the manner described by Ney, since it would be difficult to mobilize the short proximal end.

Lesions of the facial nerve below the stylomastoid foramen due to trauma lend themselves to anastomosis or neurolysis, provided the injury has occurred above the division of the nerve into the numerous muscular branches of the face. In cases of tumors of the facial nerve, the nerve may be preserved occasionally, and the function may return, if the

6. Wallerstein, E. U.: Case of Acute Mastoiditis Complicated by Facial Paralysis, Operation, Recovery, Virginia M. Month. **1**:615-616, 1923; ibid, Laryngoscope **34**:131-133 (Feb.) 1924.

7. Lillie, H. I.: Personal communication.

surgical technic described by Sistrunk [8] and Adson [9] in removing tumors of the parotid is employed.

Before surgical intervention, it is well to call to mind the theory of degeneration and regeneration of nerve fibers. When a nerve is divided from its cell body, the peripheral segment degenerates. The axon breaks up into granular fragments, and the myelin undergoes chemical change and forms irregular fatty globules. Later, the degenerated axon and myelin are entirely absorbed. The neurilemma cells of a degenerated peripheral nerve fiber and their cytoplasm increase, and they become united end-to-end to form nucleated protoplasmic bands or band fibers. These changes in the nerve fiber are known as wallerian degeneration.

In regeneration, new axons grow out from the old ones in the central undegenerated portion of the nerve. These grow into the distal degenerated stump and find their way along the recently formed nucleated protoplasmic bands to the terminals of the degenerated nerve. These band fibers serve as conduits for the growing axons, and from them the new neurilemma sheaths are differentiated. Thus, while the neurilemma cells and the band fibers derived from them appear to be incapable in themselves of developing new nerve fibers in the peripheral stump, they play an important part in nerve regeneration in cooperation with the new axons from the central stump.[10]

With physiologic interruption of the facial nerve there may be a lack of volitional movement of the facial muscle; when this is restored by faradic stimulation, recovery will probably take place in from six to twelve weeks, but failure of the faradic impulses to produce a response after a period of from three to six weeks, shows that degeneration has set in. If the lesion producing the paralysis is due to edema or inflammation, spontaneous regeneration may take place, and evidence of recovery should manifest itself in from three to six months after the onset of the paralysis. Cases have been known in which regeneration has resulted as long as a year after the onset of the paralysis; owing, probably, to slow recovery of the inflammatory lesion in the canal. When an injury of inflammatory origin to the nerve results in marked fibrosis and constriction, recovery rarely occurs, and surgical intervention becomes necessary, as in complete interruption of the facial nerve from trauma.

It is well to wait a reasonable length of time for spontaneous recovery before anastomosing nerves. The surgeon should not be misled by the occasional deforming contraction on the paralyzed side, but should

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8. Sistrunk, W. E.: Mixed Tumors of the Parotid Gland, Minnesota Med. **4**:155-160 (March) 1921.

9. Adson, A. W., and Ott, W. O.: Preservation of the Facial Nerve in the Radical Treatment of Parotid Tumors, Arch. Surg. **6**:739-746 (May) 1923.

10. Cajal, Ramon y: Nervenregeneration, Leipzig, J. A. Barth, 1908, pp. 196.

rely on the electrical reactions of degeneration and regeneration, bearing in mind that the faradic current should be employed in testing for them. The galvanic current will produce a myotatic reaction in a paralyzed muscle for some time after a nerve injury. Galvanic reaction is important, since little is to be expected from operation on a nerve if the muscles have become fibrotic and fail to respond. During the period of paralysis, it is well to prevent overstretching of the muscle by devices such as colored adhesive plaster to the cheek, as suggested by Frazier,[11] or a hook applied to the angle of the mouth and supported from the side of the head. Unless there is evidence of complete severance of the facial nerve, from the history of the injury, it is well to postpone nerve anastomosis for a period of six months. If there is suggestive evidence of regeneration, in the form of recovery of muscular tone at the end of six months, it is well to defer surgical interference for from three to six months longer. The same principles affecting nerve anastomosis in general, affect nerve anastomosis of the facial nerve, that is, the sooner the anastomosis is performed after the injury the better will be the result, bearing in mind, however, that the nerve should be given an opportunity for spontaneous recovery; if this does not occur within from six to nine months, it is very doubtful whether such regeneration will ever take place.

It is known that a good physiologic result will follow if the anastomosis is performed during the first six or twelve months after the injury, but that the degree of recovery diminishes progressively if operation is postponed beyond twelve months until after a period of three years, and very little healing may be expected. This is explained on the ground that fibrosis takes place in the peripheral segment and does not permit the entrance of new neurofibrillae. It is also known that the ideal anastomosis requires careful dissection, absolute control of hemostasis, and accurate apposition of the freshened ends of the nerve, with previous removal of any scar tissue or redundant fascia, and suture with fine silk (No. 00) in the epineurium, without tension on the ends of the nerve sutured. When recovery takes place spontaneously following complete degeneration of the facial nerve, it is not uncommon to find that the central nerve fibers have failed to follow the old pathway but that fibers have crossed so that the axon that previously innervated the orbicularis oris may now innervate the orbicularis palpebrarum, and in attempting to smile, the lids of the eye may close, or in attempting to close the eyelid the patient may grin.

If the hypoglossal or the spinal accessory nerve is anastomosed, improvement of muscular tone is the first sign of recovery; it reduces

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11. Frazier, C. H.: Surgery of the Head, Neck and Breast, Progr. Med. **1**: 65-67, 1919.

the flaccidity, or flabbiness, of the cheek; this may appear in from three to five months. Voluntary control of the buccinator and of the orbicularis oris is the next manifestation of improvement, then voluntary control of the other muscles of the face, and finally of the frontalis portion of the occipitofrontalis and of the depressor anguli of the lip.

When the eleventh or twelfth nerve is employed in the anastomosis, associated movements of the shoulder or tongue produce contraction of the facial muscles, and it is not until the cortical centers have become reeducated that the patient is able to produce symmetrical movements

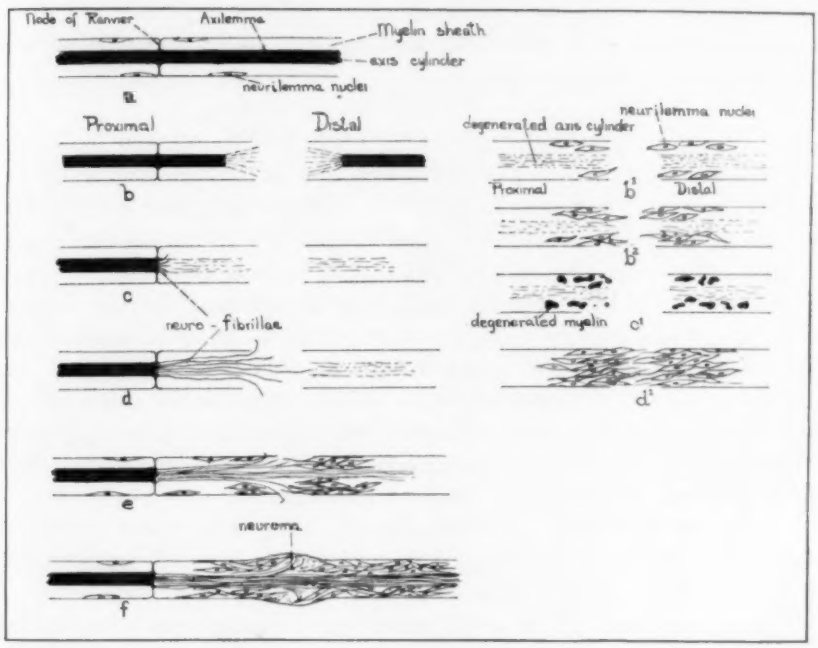

Fig. 1.—*a*, a sketch of a normal medullated nerve illustrating the axis cylinder, axilemma, myelin sheath, neurilemma (Schwann sheath) and its nuclei, and the node of Ranvier. *b*, abortive regeneration of the axis cylinder brought out by the pyridin silver stain (Cajal) on the third day following severance of the nerve. *c*, wallerian degeneration of axis cylinders which begins on the third day and is quite complete on the eighth day following severance of the nerve brought out by pyridin silver stain. *d*, regenerative process of the axis cylinder with an increase in number of fibrillae. *e*, sketch illustrating the process of regeneration in which the neurofibrillae from the axis cylinder entered the distal segment following protoplasmic bands through the intervening gap (twenty-eight days after operation). *f*, regeneration of the axis cylinder with the formation of the reticulum of the myelin sheath (thirty-five days after operation). b^1, the proliferated changes that have taken place in the Schwann sheath nuclei on the third day following severance, brought out by hematoxylin and eosin method. b^2, progress of the proliferative change in Schwann sheath cells on the eighth day following severance of the nerve. c^1, wallerian degeneration of the myelin sheaths on the eighth day brought out by Marchi's method. d^1, bridging of the intervening gap with protoplasmic bands arising from Schwann sheath nuclei.

or to express emotion. In view of the incoordinate movements of the face, the associated movements of the face with the shoulder in the spinofacial, and the associated movements of the tongue in the facio-hypoglossal anastomosis, the surgeon desires, if possible, to anastomose the severed ends of the facial nerve. When compelled to employ an adjacent nerve, he must first consider the disadvantages of sacrificing the nerve employed, and then compare the results of regeneration with the possibility of reeducation; plastic operation on the muscle should not be employed unless it is impossible to get results from nerve anastomosis, when, for example there is an injury across the face, making anastomosis of the peripheral branches on the cheek or eyebrow impossible, or when paralysis has been of such long standing that nerve regeneration will not take place (Fig. 1).

REVIEW OF THE LITERATURE

The suturing of a divided nerve has been in practice apparently for centuries, since Guy de Chauliac,[12] in a work compiled about 1363, quoted from Avicenna and others the statement that divided nerves and tendons should be sutured, and said that he himself had heard of many instances in which recovery had been complete following suture. Flourens [13] was the first to carry out experiments in nerve anastomosis. He divided the two main nerves of the brachial plexus in a cock and then transposed the proximal end of the one nerve to the distal end of the other and vice versa, and reported that regeneration was complete in two months. Kennedy [14] performed similar experiments on the fore limb of dogs, crossing flexor and extensor nerves.

Létiévant,[15] in 1873, suggested nerve grafting as a possible expedient when the ends of a divided nerve could not be brought together, but preferred an autoplastic flap procedure.

Sick and Sänger,[16] in 1893, joined the proximal end of a divided musculo-spiral nerve to the median nerve by a bridge formed of half the thickness of the median nerve.

In 1895, Ballance [17] united the facial to the spinal accessory nerve, but did not report the case until 1903. Restoration of the normal

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12. Chauliac, Guy de: Quoted by Ballance.

13. Flourens, P.: Quoted by Ballance.

14. Kennedy, Robert: Quoted by Cushing.

15. Létiévant, J. J. E.: Traité des sections nerveuses; physiologie pathologique, indications, procédés operatoires, Paris, Baillière, 1873.

16. Sick, C., and Sänger, A.: Heilung einer in Folge traumatischen Defects bedingten Lähmung des Radialis durch Vernähung des peripheren Endes dieses Nerven mit dem Medianus, Arch. f. klin. Chir. **54**:271-279, 1897.

17. Ballance, C. A.; Ballance, H. A., and Stewart, P.: Remarks on the Operative Treatment of Chronic Facial Palsy of Peripheral Origin, Brit. M. J. **1**: 1009-1013, 1903.

electrical reactions and of the nutrition of the muscles of the face was obtained, together with associated, but no dissociated, movements. He believed that he was the first to perform this operation. However, he quotes Bevers who states that anastomosis of the peripheral end of the facial to the central end of another cranial nerve was first performed in 1879. This statement is based on a passage in an article by Bronislaus Sawicki[18] which reads: "T. Drobnik, 1879, unpublished observation, patient, a man, facial palsy due to suppuration in the petrous. The

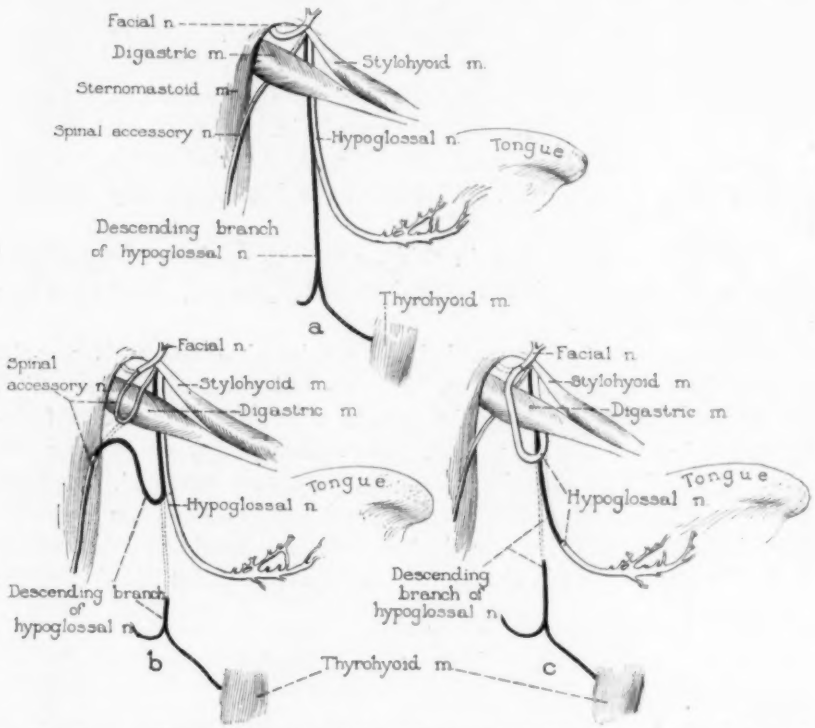

Fig. 2.—*a*, schematic drawing of the spinal accessory, hypoglossal, and descendens hypoglossi with their relation to muscles. *b*, spinofacial anastomosis with an anastomosis of the descendens hypoglossi to the distal segment of the spinal accessory to prevent atrophy of the sternocleidomastoid and trapezius muscles. *c*, faciohypoglossal anastomosis and anastomosis of the descendens hypoglossi to the distal segment of the hypoglossal nerve to prevent atrophy of the tongue. Anastomoses *b* and *c* represent the accepted nerve anastomosis for facial paralysis when it is impossible to anastomose the nerve within the facial canal.

peripheral end of the facial was united with the central end of the external branch of the spinal accessory. Result at first nil, but after several months it was ascertained that the features had become more symmetrical."

18. Sawicki, Bronislaus: Quoted by Ballance.

In 1898, Furet suggested to Faure that facial palsy might be cured by anastomosing the facial with the hypoglossal nerve; Faure,[19] however, preferred the spinal accessory, and performed the operation in January, 1898. Following Faure's first case came the experimental work of Barrago-Ciarella,[20] who divided the facial nerve in dogs and sutured it to the spinal accessory, with evidence of beginning recovery in four months and complete recovery in from six to eight months.

In the same year, Manasse,[21] working in Munk's laboratory in Berlin, divided the facial nerve at its exit from the skull in five animals and made an end-to-side suture between the stump of this nerve and the spinal accessory. His experiments were not so conclusive as those of Barrago-Ciarella, but demonstrated the passing of new axis cylinders through the anastomosis by histologic methods.

In 1903, Gluck [22] also anastomosed a paralyzed facial nerve with another nerve, and said that he felt he was entitled to priority in regard to the operation.

Cushing [23] was the first in this country to perform a spinofacial anastomosis; the operation was performed May 12, 1902, and the case was reported in 1903.

The first recorded case of faciohypoglossal anastomosis was by Körte,[24] in 1903. The operation had been performed on Dec. 20, 1901. Frazier and Spiller [25] published a preliminary report of a case of faciohypoglossal anastomosis in 1903. Since then, many cases of spinofacial and faciohypoglossal anastomoses have been recorded. In 1904, Taylor and Clark [26] reported three cases of anastomosis of the facial and hypoglossal nerves with an implant of the distal portion of the seventh nerve into an incision in the hypoglossal nerve, without division of the

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19. Faure, J.-L., and Furet, F.: Traitement chirurgical de la paralysie faciale consécutive á un traumatisme intra-rocheux; l'anastomose du facial et de la branche trapezienne du spinal, Gaz. d. hôp. **71**:259-261, 1898. Faure, J.-L.: Discussion, Rev. de chir. **36**:564-565, 1907.

20. Barrago-Ciarella, O.: La sutura dell' accessori di villis col facciale nella paralisi dei facciale, Policlinica (sez. chir.) **8**:124-139, 1901.

21. Manasse, Paul: Ueber Vereinigung des N. facialis mit dem N. accessorius durch die Nervenpfropfung (Greffe nerveuse), Arch. f. klin. Chir. **62**:805-834, 1900.

22. Gluck: Ueber Nervenplastik, insonderheit die Greffe nerveuse bei Facialislähmung, Centralbl. f. Nervenh. u. Psychiat. **26**:404-409, 1903.

23. Cushing, Harvey: The Surgical Treatment of Facial Paralysis by Nerve Anastomosis, with the Report of a Successful Case, Ann. Surg. **37**:641-659, 1903.

24. Körte, W.: Ein Fall von Nervenpfropfung: des Nervus facialis auf den Nervus hypoglossus, Deutsch. med. Wchnschr. **29**:293-295, 1903.

25. Frazier, C. H., and Spiller, W. G.: The Surgical Treatment of Facial Palsy; Preliminary Report of One Case, Penn. M. J. **7**:476-480, 1903-1904.

26. Taylor, A. S., and Clark, L. P.: The Surgical Treatment of Facial Palsy, with the Technic of Facio-Hypoglossal Nerve Anastomosis, Med. Rec. **65**:321-325, 1904.

hypoglossal. Ballance,[27] in 1909, described a case, in which, after anastomosing the facial to the hypoglossal nerve, he joined the distal segment of the hypoglossal to a strip of the spinal accessory nerve, and quoted Sargent,[28] who reported five cases of faciohypoglossal anastomosis performed by himself, in two of which he anastomosed the peripheral end of the hypoglossal to a slip taken from the spinal accessory nerve for paralysis of the tongue, but did not obtain recovery from this in either instance.

Grant,[29] in 1910, reported a double anastomosis, a spinofacial anastomosis and an anastomosis of the descendens noni, 1 inch below its point of separation from the hypoglossal, to the peripheral end of the divided

Fig. 3.—Spinofacial anastomosis with a cuff of vein from the external jugular, as suggested by Beckman. This covering is not necessary but aids in keeping the anastomosed ends in proper apposition, avoiding tying the sutures too tightly at the point of anastomosis.

spinal accessory, with beginning recovery of the facial nerve in three and one-half months, and satisfactory recovery in nine months. There

27. Ballance, C. A.: A Case of Facial Palsy Treated by Faciohypoglossal Anastomosis in which an Anastomosis Was Also Made Between the Spinal Accessory and the Distal Segment of the Divided Hypoglossal Nerve in Order to Prevent Permanent Lingual Paralysis and Atrophy, Lancet **1**:1675-1677, 1909.

28. Sargent, P., and Greenfield, J. G.: An Experimental Investigation of Certain Materials Used for Nerve Suture, Brit. M. J. **2**:407-410 (Sept. 27) 1919.

29. Grant, W. W.: Traumatic Facial Paralysis; Anastomosis of Facial Nerve to Spinal Accessory, and the Peripheral End of Accessory to the Descendens Hypoglossi, J. A. M. A. **56**:1438-1442 (Oct. 22) 1910.

was no resultant discomfort or weakness in function around the shoulder, and no atrophy of the trapezius muscle. Ballance,[30] in 1913, also introduced a double anastomosis, a faciohypoglossal and a descendens noni anastomosis to the peripheral portion of the hypoglossal. Alt,[31] of Vienna, described a method of opening the facial canal directly, to relieve compression on the nerve. Ney,[4] in 1922, presented an anatomic study on dissection of the facial nerve and recommended repair of the nerve in the course of the canal rather than spinofacial or faciohypoglossal anastomosis because of the dissociated movements that are prone to develop after either of the latter operations (Figs. 2 and 3).

When facial paralysis is of such a character or of such duration that nerve anastomosis seems inadvisable, numerous plastic operations, which have been described, may be considered. Eden,[32] in 1911, advised transferring a strip of the masseter muscle from its attachment on the end of the jaw to the angle of the mouth in addition to transferring a strip of temporal muscle to the outer canthus of the eye. Busch,[33] in 1910, advocated the use of an aluminum-bronze suture, attaching it to the periosteum of the zygoma and bringing it down to the angle of the mouth by means of a long needle which was again returned over the zygoma (Fig. 4). Ballance,[2] in 1919, suggested and performed an operation combining the Eden and Busch methods, but he used a platinum wire instead of a bronze one. Fenwick,[34] in 1919, recommended transplants of temporal muscle and fascia to the orbicularis oris and to the outer canthus of the eye. Gersuny,[35] in 1916, transplanted paralyzed muscle into normal muscle in the hope of transferring the innervation of the normal muscle, and reported some improvement.

From this review of the literature, it is evident that the spinofacial or the faciohypoglossal anastomosis does not offer all that is hoped for in the restoration of facial control. It is true that regeneration takes place, that facial tone returns, and that voluntary movements can again be performed, but it is also true that the patient has difficulty in expressing emotion on the side that was paralyzed and that dissociated movements of the face occur when the shoulder or the tongue is moved. The

30. Ballance, C. A.: Case of Facial Palsy, Following a Mastoid Operation, Treated by Nerve Anastomosis, Proc. Roy. Soc. Med. (Clin. Sect.) **7**:33-34, 1913-1914.

31. Alt, F.: Ein Beitrag zur operativen Behandlung der otogenen Fazialislähmung, Wien. klin. Wchnschr. **19**:1285-1289, 1906.

32. Eden, Rudolf: Ueber die chirurgische Behandlung der peripheren Facialislähmung, Beitr. z. klin. Chir. **73**:116-126, 1911.

33. Busch, H.: Kosmetische Besserung der durch Fazialislähmung bedingten Entstellung, Ztschr. f. Ohrenh. **68**:175, 1913.

34. Fenwick, G.: Surgical Treatment of Facial Paralysis, Brit. M. J. **2**:700-701 (Nov. 29) 1919.

35. Gersuny, R.: Die Operation eines Muskelauschlusses wegen Fazialislähmung, München. med. Wchnschr. **63**:719, 1916.

most ideal anastomosis is the repair of the facial nerve, that is, end-to-end anastomosis of the proximal and distal ends of the facial nerve, but even with this operation recovery is not always perfect, since spontaneous regeneration following lesions similar to Bell's palsy may result in crossed innervation (Fig. 5). Plastic operations should be resorted to when a nerve anastomosis is inadvisable or impossible.

FIG. 80.

FIG. 81.

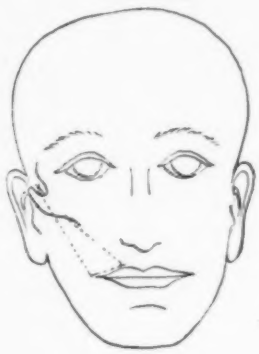
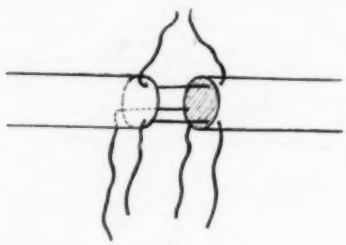


FIG. 82.

Fig. 4.—Eden's method (80). A strip of the masseter is detached and sutured to the angle of the mouth. Similarly a strip of the temporal is detached and sutured to the lower eyelid. The dotted lines show the incisions employed. The needle used by Busch (81). Busch's method (82). The dotted line indicates the passage of the wire in the tissues of the cheek from the zygoma to the outer part of the upper lip (from Ballance).



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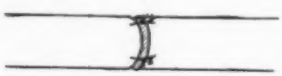

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Fig. 5*a* and a^1.—End-to-end suture with interrupted sutures of fine silk (oo) in the epineurium without tension or covering. Accepted by most surgeons as ideal anastomosis.

The problems that present themselves are spinofacial versus facio-hypoglossal anastomosis, end-to-end suture versus end-to-side suture, as suggested by Ballance, and end-to-implant suture, as suggested by Taylor. Also, is it necessary and advisable to anastomose the descending hypoglossal to the peripheral end of the hypoglossal or to the peripheral end of the spinal accessory nerve, and are plastic operations worth while?

REVIEW OF CASES

Before presenting the arguments for and against these various operations, I wish to present the results obtained in a series of thirty cases in which various operative procedures for facial paralysis were employed. For convenience of study, the cases are divided into seven groups, according to etiology: Group 1, one case, due to the development of a tumor at the angle of the jaw; Group 2, five cases, due to a parotid tumor; Group 3, three cases, due to the wound of an incision to drain an abscess below the ear; Group 4, one case, due to fracture of the temporal bone; Group 5, three cases, due to a bullet wound; Group 6, one case, due to mastoiditis and not relieved by radical mastoid operation, and Group 7, sixteen cases, following mastoid operation.

Group 1.—The patient in this group was a child, aged 21 months, with a history of facial paralysis of fourteen months' duration. The tumor was explored, found to be a desmoid in the posterior belly of the digastric muscle, and removed. The facial nerve was then exposed and found to be so much compressed by the bony tumor of the muscle that a simple neurolysis was deemed insufficient, and therefore this area was resected and the peripheral segments anastomosed to the proximal portion of the spinal accessory nerve. Two weeks later the child was dismissed from the clinic after an uneventful convalescence, and one hundred seventy-three days after operation the parents reported the return of muscular tonus and movements in the face associated with shoulder movements. Improvement continued for a year, and the results have been very satisfactory, there being some improvement in the control of expression (Fig. 6).

Group 2.—In the five cases in this group, I attempted to relieve or repair the facial nerve in the presence of a malignant tumor of the parotid gland. With the exception of one case, the results were very unsatisfactory. Four different types of operation were performed in these cases. In one, a neurolysis was performed, that is, removal of the tumor after dissection of the facial nerve. This procedure is warranted in selected cases; the technic of the procedure is described in a separate paper.[9] In four months, the patient had completely recovered from the facial paralysis, and at the time of writing, three years after operation, she is well. In another case in this group, an attempt was made to anastomose the peripheral segments of the facial nerve in the region of the parotid gland; both the operative and the postoperative results were unsatisfactory, as the fusion method of anastomosis had to be relied on. Only slight regeneration occurred in but a few of the nerve fibers. In two cases a spinofacial anastomosis was performed, since the nerve had been stretched by the parotid tumor to such an extent that no regeneration

occurred after removal of the tumor. In these cases the seventh nerve was explored and identified and then anastomosed to the spinal accessory nerve. Signs of regeneration appeared in one case one hundred eighty-three days after operation, and, in the other, one hundred five days after operation. In the first, improvement continued for six months, and in the second for nine months, with reasonable restoration of control in the facial muscles, when there was evidence of recurrence of the parotid tumor and consequent progression of the facial paralysis. The other patient in this series had had an extensive, mutilating operation in the parotid region, which precluded the possibility of a nerve anastomosis; therefore a plastic operation was performed according to the technic of Eden, the anterior portion of the masseter muscle being transferred to

Fig. 6.—Left facial paralysis of fourteen months' duration due to a desmoid tumor of the posterior belly of the digastric muscle. Photograph taken six weeks after operation (*a*). The patient six months after operation (*b*). There is marked improvement in muscular tone with some movement of the face.

the angle of the mouth and sutured to the orbicularis oris by interrupted silk sutures, and a strip of temporal muscle being sutured to the outer canthus of the eye. The result was unsatisfactory; suppuration developed around the silk sutures, which had to be removed, and the patient soon developed an intracranial extension of the carcinoma and died from metastatic carcinoma, twenty-eight months after her admission to the clinic.

Group 3.—In one of the three cases in this group, in which facial paralysis had existed for ten months following drainage of a parotid abscess without any evidence of regeneration, spinofacial anastomosis was made. Six months later it was reported that there was no improvement; at present, eighteen months after operation, the patient cannot be located. The second patient had had facial paralysis of fourteen years' duration, as the result of drainage of a suppurative cervical gland in

the region of the parotid. A plastic operation of the Eden type was advised and performed, but the results were unsatisfactory. The third patient was a woman, aged 37, who had suffered from facial paralysis for thirty-six years, beginning after drainage of suppurative cervical glands in the region of the parotid. In view of the long duration of the paralysis and the atrophy of the muscles, nerve anastomosis was considered inadvisable, and the Eden plastic operation was performed, that

Fig. 7.—The patient had had right facial paralysis for thirty-six years. Photograph taken March 11, 1922 (See Figs. 8 and 9).

is, the anterior third of the masseter muscle was loosened from its attachment on the mandible through an incision under the angle of the jaw, carried through a tunnel of subareolar tissue to the angle of the mouth, and sutured to the old orbicularis oris muscle and the neighboring fascial tissue. This was done through a second incision at the angle of the mouth. The temporal muscle was not attached to the orbicularis palpebrarum; instead, the outer canthus of the eye was narrowed by a suture, and while the result was far from perfect, it was well worth while. This patient has learned to smile on the paralyzed side by

moving the masseter muscle, as one would in bringing the teeth forcibly together; she has also learned to smile on opening her mouth, again stimulating the masseter muscle to contract; the length of the masseter flap prevents the paralyzed side from moving across the median line to the normal side as it did previous to operation (Figs. 7, 8 and 9).

Group 4.—The one patient in this group was a strike-breaker who, on returning from work, was hit by an iron bar across the mastoid area

Fig. 8.—Twenty days after transference of the anterior half of the masseter muscle to the paralyzed orbicularis oris. A small scar and slight depression may be seen at the point of suture in the region of the orbicularis oris. The patient is able to raise the right angle of mouth slightly by bringing the masseter muscle into play; this is due to preservation of the nerve supply to the masseter muscle.

during an encounter. The paralysis lasted four months, without any signs of regeneration. The usual spinofacial anastomosis was performed; signs of improvement in motor control appeared one hundred twenty-four days after operation, and improvement continued for fourteen months. During this time he remained in Rochester and was employed as a laborer in the construction of the new wing at St. Mary's

Hospital, so he was observed very closely. He was a young man, aged 27, who was very particular about his appearance; he cooperated eagerly in the postoperative treatment, which consisted of massage, occasional galvanic treatment, and especially of exercise of the facial muscles. He was instructed to spend two periods daily in front of a mirror, in an effort to develop not only movements of the face in conjunction with movements of the shoulder, but also coordinated

Fig. 9.—Thirty-one months after operation. Considerable improvement is shown. Patient has learned to elevate the angle of the mouth with the use of the masseter muscle. Outer canthus of right eye narrowed by suture.

movements on both sides of the face, as well as to inhibit the movements of the face when moving the shoulder. This patient left Rochester fourteen months after operation, with a most satisfactory return of tonus, facial control and a fair recovery of emotional expression. Greater restoration of function could scarcely have been asked for from a nerve anastomosis (Figs. 10, 11 and 12).

Group 5.—In one case of this group, in which paralysis was due to injury by a bullet, the wound was below the stylomastoid foramen.

presenting a resultant contraction and scarring. A neurolysis was performed. Considering that the paralysis had existed for forty-eight months at the time of operation, the improvement was marked. The first signs of recovery were noted one hundred eighty days after operation, and there was a continuance of improvement for two years and three months. The result obtained in this instance is interesting, especially because of the long duration of the paralysis and the slowness of the recovery. The other two patients in this series had sustained

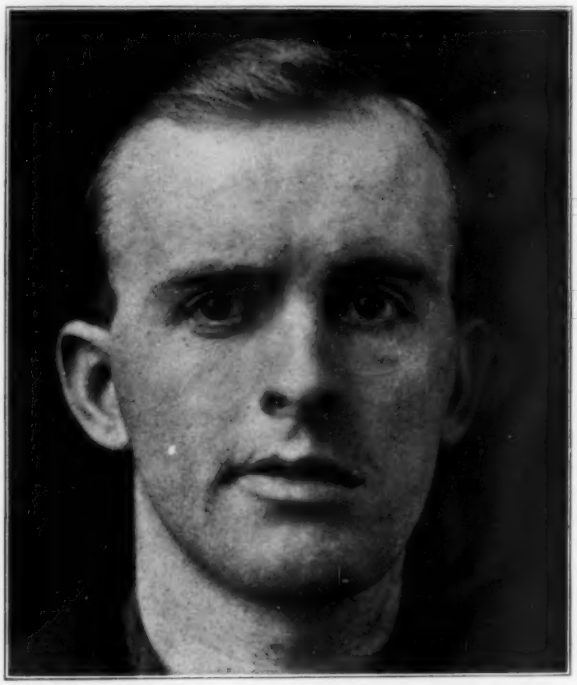

Fig. 10.—Left facial paralysis of five months' duration, due to injury to the temporal bone. Photograph taken ten weeks after spinofacial anastomosis (See Figs. 11 and 12).

bullet wounds interfering with the spinal facial canal, with an average duration of paralysis of three and one-half months. Spinofacial anastomoses were performed; one patient had not improved at the end of three hundred seventy days, the other was not heard from.

Group 6.—The patient in this group was an Indian girl, aged 14, who had had facial paralysis for thirteen weeks, preceded by otitis media of tuberculous origin involving the mastoid. She had also had Pott's disease. A radical mastoid operation was performed, but did not relieve the paralysis; later a spinofacial anastomosis was made. The

first signs of regeneration were noted one hundred forty days after operation; muscular tone appeared ninety days after operation. Improvement continued for fifteen months, and there was a satisfactory recovery of movement in the face; the patient was, however, unable to dissociate the facial movements from the shoulder movements, and there was no return of motor function in the frontalis muscle. She died from tuberculous meningitis twenty-eight months after operation.

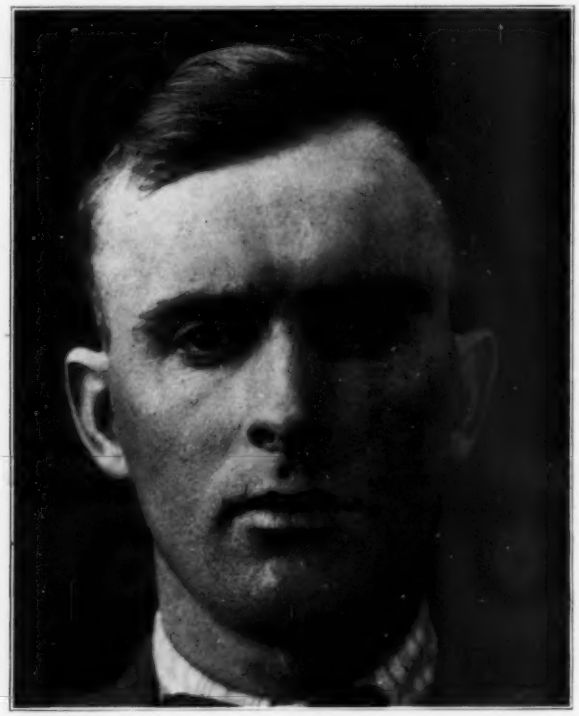

Fig. 11.—Sixteen months after operation. There is evidence of recovery of muscular tone and narrowing of the palpebral angle.

Group 7.—The average duration of paralysis before surgical intervention in thirteen of the cases in this group, in which the paralysis followed a radical mastoid operation, was six and one-third months; in the remaining three cases the condition was of varying duration. In one case it had lasted for nine years, in another for ten years; operation was performed in each case more than four years ago, but neither have reported to us. No doubt the result in each case is a failure, as the paralysis was of such long standing. The other one of the three patients said that the paralysis had lasted five years; it was somewhat surprising that motor regeneration began two hundred sixteen days after

operation, and improvement in muscular tone a month or two prior thereto, the latter continuing for eight months. The degree of improvement was graded as better than 3 + on the basis of 1 to 4; there was good motor control of the face, but no improvement in the frontalis muscle, and some difficulty in dissociating the shoulder and facial movements.

The average return of motor function in the face occurred one hundred ten days after operation in this group. It was, however,

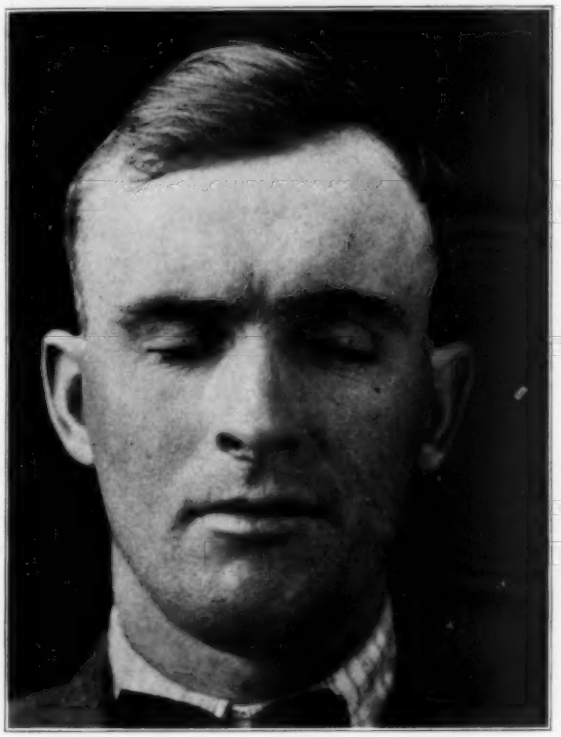

Fig. 12.—Patient in repose sixteen months after operation. Almost complete closure of the eye without elevation of shoulder or any serious contractions of the face is shown. There has been some return of movement in the left half of the frontalis muscle also. This patient has progressed very rapidly in dissociation of the various movements of the face and eye.

associated with movements in the shoulder. Muscular tone invariably returned a month or six weeks before motor function, and improvement continued for fourteen months. Five of this group have acquired reasonable control of the facial movements without associated movements of the shoulder, although there would be a suggestion of associated movement in the face if the shoulder and arm were elevated to a perpendicular position. Three other patients of this group had about

a 75 per cent. return of motor control in the face. One patient regained partial control, equivalent to about 50 per cent. The reports received from two were so meager that the degree of recovery could not be determined. Four patients did not reply to our letter of inquiry, and one patient was operated on two months ago, too recently to expect any return of motor control (Figs. 13, 14, 15, 16, 17, 18 and 19).

Fig. 13.—Right facial paralysis of nine months' duration, developing subsequent to a mastoid operation (See Figs. 14, 15, 16, 17, 18 and 19).

SUMMARY

Faure,[36] in 1898, was the first to suggest the faciohypoglossal anastomosis; however, the procedure was not employed at that time, but was first carried out by Körte in 1901. Since the work of these two men, there have been numerous ardent supporters of this operation. Ballance later favored the faciohypoglossal anastomosis, although he was the first to perform a spinofacial anastomosis (Oct. 12, 1895).

36. Faure, J.-L.: Traitement chirurgical de la paralysie faciale par l'anastomose spino-faciale, Assoc. franç. de chir., Proc. verb. **12**:190-195, 1898; ibid, Semana méd. **18**:426, 1898.

Other supporters of the faciohypoglossal anastomosis are Taylor, Frazier, Spiller, Gibson, and Coleman,[37] who give very good reviews on the subject. Ballance, in explaining the reason for his change, states that one operation is not more difficult than the other, but that the hypoglossal anastomosis is to be preferred because the cortical center for the movements of the face is more closely associated with that for the tongue than with that for the movements of the shoulder, and because

Fig. 14.—Six months after operation. Patient attempting to smile without moving the shoulder. There is evidence of marked improvement in muscular tone and in ability to close eye.

paralysis of half of the tongue is much less inconvenient than paralysis of the sternomastoid and trapezius muscles. The cortical centers for the face and the tongue overlap, and one would therefore anticipate that dissociated facial movements would be greater when the nerve was connected with the tongue center than when the connection was made with the shoulder center, which is much farther off. Moreover, it may

37. Coleman, C. C.: Surgical Treatment of Facial Paralysis, Virginia M. Month. **49**:180-188 (July) 1922.

be recalled that the movements of the tongue are closely associated with those of the lips, so much so that in health the orbicularis oris and the transverse fibers of the tongue contract together. It is more than probable that the facial nerve derives its lip fibers from the hypoglossal nucleus. There is, therefore, a close relation, not only between the cortical face center and tongue center, but also between the facial and hypoglossal nuclei in the bulb. Ballance states further that regeneration is slower to occur following spinofacial anastomosis than following

Fig. 15.—Lateral view, showing lack of muscular tone and widening of the palpebral angle.

hypoglossal anastomosis. Experience has shown that atrophy of half of the tongue leads to no material inconvenience in most cases, but that loss of power in the sternomastoid and trapezius muscles may be associated with deformity, discomfort, inconvenience and even worse results.

Coleman, in arguing for faciohypoglossal anastomosis, says that it is preferable to spinofacial anastomosis, since the results of the former come nearer to simulating the normal functions of the face. He enumerates the objections made to this form of anastomosis as: atrophy

and deformity of the tongue, difficulty in mastication, swallowing and speech, and the complaint of a most persistent and troublesome type of associated movements, and says that the first two objections may be dismissed because the deformity of the tongue is of no moment, and any interference with the function of the tongue immediately after section of one hypoglossal nerve soon disappears. Some patients seem to attribute the collection of food in the paralyzed cheek to unilateral paralysis of the tongue. This inconvenience disappears with the resump-

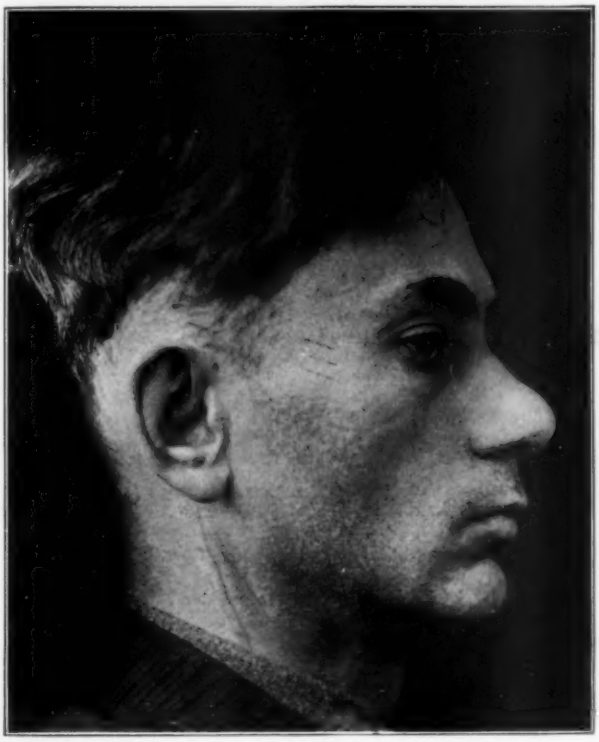

Fig. 16.—Lateral view after operation, showing return of muscular tone and narrowing of the palpebral angle.

tion of the buccinator function, to the loss of which it is mainly due. It is no doubt true that in some cases the associated movements resulting from faciohypoglossal anastomosis are conspicuous and persistent.

Gibson argues for the faciohypoglossal anastomosis on the ground that the tongue is used as a medium of speech in emotional expression.

While the pendulum has apparently swung in favor of the faciohypoglossal anastomosis, much is to be said in favor of the spinofacial type. My experience has been with the spinofacial anastomosis, since it was the operation advocated and employed by my predecessor, Beck-

man.[38] Aside from studying the results obtained from spinofacial anastomosis, it was my privilege to study a case two years after faciohypoglossal anastomosis had been performed. It is true that the cortical centers of the seventh and twelfth nerves are closer than those of the seventh and eleventh nerves; it is also true that the bulbar centers of the seventh and twelfth are closer than those of the seventh and eleventh, and that the hypoglossal nerve in aiding speech acts as a nerve of expression, but in comparing the clinical results I question whether the anatomic explanations substantiate the clinical findings. Ballance,[39] who performed the first spinofacial anastomosis,

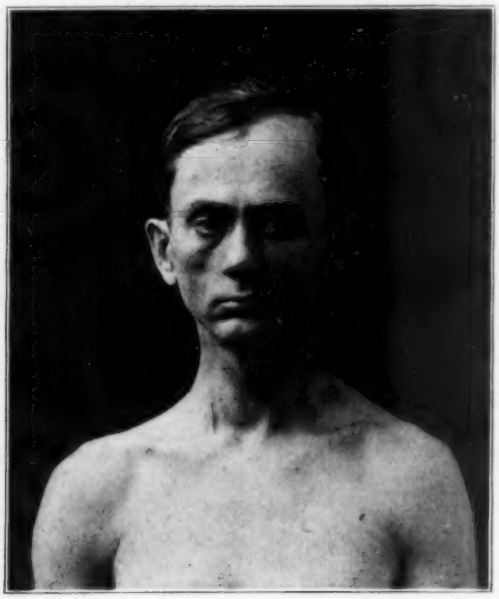

Fig. 17.—Thirty-eight months after operation, showing patient while smiling, without conscious attempt to smile on the right side.

reported several years later when he again had occasion to see the patient that there was a restoration of the normal electrical reaction and of the nutrition of the muscles of the face, together with associated movements, but that there were no dissociated movements. Cushing,[23] in reporting the case of his patient in 1903, operated on in 1902, stated that while some facial asymmetry persisted, there was marked improvement and some coordination of expressional movements, that the patient

38. Beckman, E. H.: Surgical Treatment of Facial Paralysis, J. Mich. State M. Soc. **13**:681-685, 1914.

39. Ballance, C. A.: Results Obtained in Some Experiments in Which the Facial and Recurrent Laryngeal Nerves Were Anastomosed with Other Nerves, Brit. M. J. **2**:349-354 (Aug. 30) 1924.

at that time was able to dissociate movements of the eye, nose and mouth slightly, and that he had considerable facial movement without shoulder action. In another report, two hundred seventy days postoperatively, he stated that electrical reactions were found to be normal and equal on both sides, and that the individual coordinate movements were under much better control than at the last examination. The eye could be kept almost completely closed without effort, and the attempt to whistle showed comparatively little difference in the contraction of the two halves of the buccinator. He stated further that volitional control had

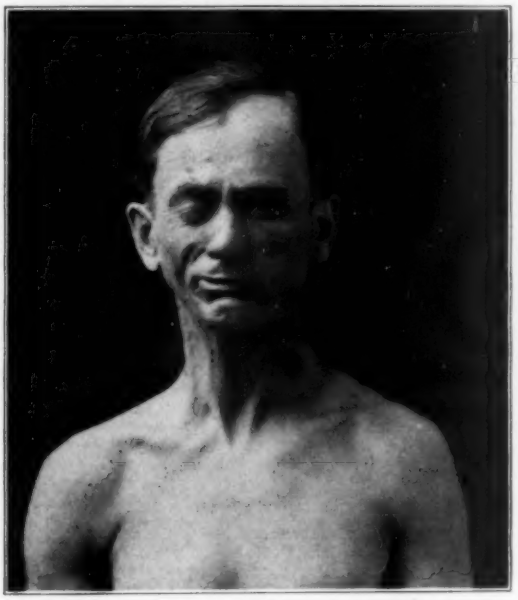

Fig. 18.—Thirty-eight months after operation, showing patient forcibly contracting muscles of face and muscles of eye without movement of shoulder. Evidence of control of facial muscles without associated movements of shoulder is shown.

returned in the individual group of muscles, and that it could be effected without associated shoulder movement or contraction in other facial muscles. However, emotional expression failed to improve in a corresponding degree, and considerable asymmetry persisted.

Cushing,[40] in a personal communication, states that he has performed a great number of spinofacial anastomoses since those reported in 1907, some of them with brilliant results, with what would appear to be

40. Cushing, Harvey: Remarks on the Surgical Treatment of Facial Paralysis of Trigeminal Neuralgia, with Exhibition of Patients, Tr. Am. Surg. A. **25**: 275-283, 1907.

emotional control, although he believes that emotional control is perhaps more a matter of training.

My own experience with spinofacial anastomosis has shown that regeneration is very satisfactory, and that in some cases there is evidence of emotional control. One patient, particularly, made very rapid progress (the patient referred to earlier who had sustained a fracture of the temporal bone). This man was able to express emotion, and there was coordination of the facial movements, which I attribute to reeducation.

With reference to the disadvantages of the paralysis of the trapezius and sternocleidomastoid muscles, there is considerable atrophy, it is true, and at first some tenderness and pain; however, as the patient's convales-

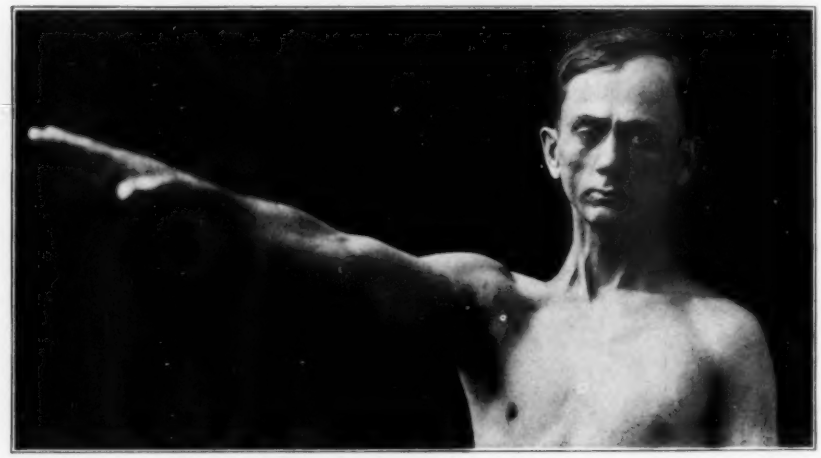

Fig. 19.—Thirty-eight months after operation, showing ability to use arm without discomfort or movements of the face.

cence progresses, there is little complaint of discomfort. It must also be borne in mind that a spinal accessory injury is a very common occurrence in a complete dissection of the posterior triangles for tuberculous glands, and that the sternocleidomastoid muscle is frequently sacrificed with a complete dissection of the neck for malignant glands; aside from their atrophy or absence, the patient rarely complains of any discomfort or lack of function. So far as the functional results are concerned, the patient is well able to carry on all of his regular activities. Should the patient be a girl and desirous of wearing low cut dresses, there would be a decided disadvantage, but in the case of a man, the atrophy is very readily covered. On the other hand, it is quite difficult to cover the tongue at all times to avoid showing the atrophy. Certainly, the inconvenience in chewing, swallowing, and speech is no greater than

the inconvenience entailed in the trapezius and sternocleidomastoid muscles.

So far as the completeness of regeneration is concerned, it is difficult to state which procedure is the more effective. I believe that the results are about equally good, but I have employed the spinofacial method because of the discomfort and the exaggerated associated movements that occur during the regenerative period which are apparently worse after faciohypoglossal anastomosis, since the patient uses the tongue more frequently than the shoulder. Surgeons who advocate faciohypoglossal anastomosis frequently insinuate that it is embarrassing for a young lady to carry a parasol while strolling because of the associated movements between the shoulder and the face, suggesting flirtation with passers-by, but this can be avoided if she will but carry the parasol in her other hand. In advocating faciohypoglossal anastomosis, one should not forget that it is perhaps much more embarrassing, following this procedure, to make grimaces and wink constantly when speaking to friends or eating in their presence. Following a spinofacial anastomosis, one can gently shrug the shoulder to produce the associated smile when desired.

The arguments on both sides have been overdrawn, perhaps, but it is obvious that both spinofacial and faciohypoglossal anastomoses are justified if it is impossible to anastomose the severed ends of the facial nerve; the latter procedure should be performed, of course, when possible (Cushing). The degree of improvement is probably equally good in spinofacial and in faciohypoglossal anastomoses. Paralysis of the tongue may be avoided in the faciohypoglossal anastomosis by transference and suture of the descendens hypoglossi to the distal end of the hypoglossal, as suggested by Ballance. Atrophy of the sternocleidomastoid and of the trapezius muscles in the spinofacial anastomosis may be avoided by anastomosis of the descendens noni into the distal end of the spinal accessory, as suggested by Grant.

End-to-end suture with complete division of the nerve is perhaps more satisfactory than the employment of only half of the nerve, as it is more difficult to dissociate the movements of the facial nerve from those of the spinal accessory or the hypoglossal as the nerve regenerates. Taylor has, it is true, obtained some very good results by implanting the distal end of the facial nerve into a slit in the hypoglossal nerve, and Ballance has obtained satisfactory results from end-to-side anastomosis. The results obtained in the two latter instances must have been due to the cutting of axons during the process of grafting which, as they regenerated, entered the distal segment of the facial nerve. End-to-end anastomosis with fine sutures of silk in the epineurium, without tension, is the accepted type of anastomosis.

Plastic operations, as a whole, are unsatisfactory, since the patient develops very little voluntary control and the most that can be hoped for is a diminution in the deformity.

CONCLUSIONS

1. Inflammatory lesions of the facial nerve, producing facial paralysis, should be given a reasonable length of time to recover spontaneously, but if there are no signs of recovery in six months, some type of nerve anastomosis is indicated.

2. The ideal nerve anastomosis is the end-to-end suture of the involved nerve, below the canal as well as in the canal, when possible. There is very little difference between faciohypoglossal and spinofacial anastomosis; the choice of operation is a personal one. The degree of improvement depends on the accuracy of the nerve anastomosis, hemostasis, the absence of tension on the sutured ends, the intelligence of the patient and the degree of reeducation that may be accomplished. To prevent atrophy of the tongue and of the trapezius and sternocleidomastoid muscles, the descendens hypoglossi may be anastomosed.

DISCUSSION

Dr. J. B. Potts, Omaha, Neb.: I wish to report a case of facial anastomosis done for us by Dr. Alfred Brown of Omaha. The anastomosis was on a patient who had had a sarcoma of the middle ear, and the facial paralysis was secondary to the sarcoma. One and a half years after the facial paralysis the patient showed no evidence of return of the sarcoma and was advised to have an anastomosis. The anastomosis was of the hypoglossal to the facial nerves and the descendens hypoglossi to the stump of the hypoglossal. It is fifteen months since the operation was performed and the results have been very satisfactory. After about three months she noticed the first effect of the anastomosis, which was a slight feeling of tenseness in her face on the application of the galvanic sponge to the cheek, but she has mastered the art of working her face until it is almost as good as the other side. So far, there is no evidence of return of the sarcoma. The technic was similar to that described before this Section by Dr. Frazier one year ago.

We have one other anastomosis which is perfect—a facial nerve paralysis following an accident. A boy chopped a piece of wire from a bale of wire and the end flew up and made a clean cut of the facial nerve. Five months afterward he came in for relief and the nerve was anastomosed by Dr. A. C. Stoker with complete recovery.

Dr. H. I. Lillie, Mayo Clinic, Rochester, Minn.: From the otologist's standpoint, the danger of a facial paralysis following any operative interference annoys the operator as well as the patient. In the performance of the radical mastoid operation the surgeon is unusually careful about injuring the facial nerve. In certain cases, it is, however, unavoidable because the nerve may be involved in a sequestrum of bone which necessarily will have to be removed. The sequestrum may be dissected but the nerve may become involved in the healing process. One sometimes encounters the facial nerve free in the mastoid process, when it may be severed at the tip. A secondary paralysis results but this usually

clears up. I feel that the region immediately in front of the eminentia pyramidalis is the most important region to be considered in avoiding injury to the facial nerve. It is in this region mentioned that infected granulations are encountered and in attempting to remove these, particularly when there is a dehiscence, the facial nerve may be injured. The same is true in the region just below and in front of the promontory of the horizontal canal. This fact must be always borne in mind in operating on a labyrinth after the method of Hinsberg and if one enters too low he is very apt to injure the facial nerve of the promontory.

The delayed secondary facial paralysis manifesting itself many hours or days after the primary operation, though disconcerting to the surgeon, usually takes care of itself in a satisfactory manner.

The repair of this facial deformity is important and offers considerable to the patient not only from the cosmetic standpoint, but from the psychologic effect. These patients are sensitive about the deformity and have a tendency to avoid social intercourse. This can be largely overcome by operative correction. The repair of these anastomoses, I think, should be undertaken by a man familiar with that kind of work. It is different work from that which the otologist is accustomed to; it is certainly more satisfactory to have the neurologic surgeon perform this operation for us in the clinic.

Dr. Leon E. White, Boston: I agree most heartily with the essayist's remarks on the facial nerve. During my last service at the Massachusetts Eye and Ear Infirmary I had six cases of facial paralysis, so that I may qualify as an expert. It may be interesting to speak of these cases.

One patient had had a radical mastoid operation. Three weeks after the operation, she developed a complete facial paralysis on the operated side, rather an unusual complication, although it frequently happens within twenty-four to forty-eight hours afterward, but not after three weeks. We thought this was probably Bell's palsy, because she had had an acute tonsillitis a day or two before the paralysis, and there was no change that we could detect in the mastoid cavity.

The second patient had chronic ear trouble with a paralysis of two or three weeks' duration. We performed a radical mastoid operation, but could find no exposure of the nerve. A few weeks after the operation the facial paralysis cleared up.

The third patient had acute middle ear trouble. It used to be our procedure at the infirmary to operate early on patients who developed facial paralysis associated with acute middle ear trouble. I never quite realized why that was done. Now in our patients with acute middle ear trouble with paralysis, we wait until the condition in the middle ear has subsided. This patient recovered without operation.

The fourth patient was a woman with an infection in the canal. The middle ear, so far as we could determine, was practically normal. The condition probably was Bell's palsy from infection. This patient also recovered without operation.

The fifth patient had a sequestrum of the entire labyrinth. He gave a history of chronic ear trouble of some years' duration with a paralysis for about one week three weeks before entering the hospital. This paralysis cleared up spontaneously. At the time of the radical operation a sequestrum approximately of the whole cochlear was found and removed. This produced traumatism of the nerve and almost complete paralysis followed. Three months later this paralysis cleared up.

I think it is an extremely valuable point to let these patients with paralysis alone, when we feel we may traumatize them and not attempt an anastomosis for

six months or a year. Dr. Green, at one time a noted surgeon of Boston, did an extremely radical procedure, but strange to say, practically all of his patients recovered spontaneously within six months or a year, and that has given me a great deal of confidence in spontaneous recovery.

The education of the patient with paralysis is important, and if a patient can be taught to admire himself in a mirror it helps in the control of the paralysis a great deal.

Dr. C. C. Coleman, Richmond, Va.: Recovery of voluntary motion after anastomosis for relief of facial paralysis is not equivalent to restoration of complete function of the facial nerve. The reappearance of emotional expression is essential to complete recovery of the nerve. The case is different in suture of peripheral motor nerves in which the return of voluntary motion is equivalent to recovery. We have used the hypoglossal nerve for the central portion of the anastomosis for facial paralysis, after the technic of Dr. Frazier, while Dr. Adson and others have obtained good results with the spinal accessory. It seemed reasonable to believe that the reeducation of facial movements could probably be more easily accomplished by using a nerve which had a functional similarity to that of the paralyzed nerve.

It has been interesting to observe the course of some of the patients who recover spontaneously after injury. We have now under observation a patient who had a bilateral paralysis of the facial nerve following mastoid infection and operation. The left side showed beginning recovery in about three months. A faciohypoglossal fusion was performed on the right side with recovery of motion. On each side there are associated movements and fibrillation of the muscles around the eyes, with absence of movements of the brow. The results are quite similar on the two sides. We have observed in a number of patients with severe paralysis with spontaneous recovery of motion, that the end-results are quite similar to those which have had a nerve fusion. It is important, however, to give a nerve ample opportunity to recover spontaneously, because in a large majority of cases spontaneous recovery is superior to the results of nerve fusion. In injury to the nerve by sharp instruments, operation should be done promptly because the distal end of the nerve may be found with an electrode even though the smaller branches of the nerve are divided. If operation is postponed, suture of the nerve is difficult and may be impossible.

Dr. T. A. Williams, Washington, D. C.: The pathology of this condition is important. Perhaps Dr. Adson did not emphasize enough that every motor nerve will regenerate invariably, unless there is some obstruction to the passage of these fibers, and it is a fact that even when there is obstruction, the nerve fibers may find their way around the obstruction and if we delay for a while there will be functional recovery. Nageotte's experiments in Paris during the war proved this emphatically.

As to the relative merits of the hypoglossal or the spinal accessory muscle, from a theoretic aspect, whether either one is nearer the center of the facial is pure theory, because we have to remember that contiguity does not necessarily mean similarity of action in the nervous system. We also have to remember that most of the important movements we are speaking of are educated movements. We have learned to use our various muscles with great facility, and that has nearly all been done by training and efficiency.

Perhaps not enough emphasis was laid on the emotional movements of the face. Dr. Coleman did bring out the fact, however, that education is an important influence even on the automatic movements growing out of emotion, and hence I should

like Dr. Adson in his concluding remarks to tell us something about how these patients' faces look when under the influence of emotion.

Dr. A. W. Adson: Dr. Williams calls attention to an important finding in his remarks on the different centers. His statement that the term "adjacent cortical centers" does not mean that they are similar in function and that one center may assume the function of the other no doubt explains the fact that regeneration in spinofacial anastomosis may be as complete as in a faciohypoglossal anastomosis, and that reeducation of the spinofacial nerve is as good as reeducation of the faciohypoglossal nerve. I doubt if either anastomosis will respond to the finer expressions of emotion, but even though this does not take place the result is well worth while, since muscular tone returns, asymmetry in repose is diminished until it is scarcely perceptible, and the patient is able to dissociate the movements in the shoulder and tongue from those in the face, and also to express a moderate degree of emotion.

THE PATHOGENESIS OF POLYPOSIS NASI*

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The conception of the pathogenesis of the nasal polyp, in the minds of the majority of rhinologists and even pathologists, is hazy. Even in standard modern textbooks, the descriptions of this condition are confusing, and in many cases, inaccurate. The time honored association of the name myxoma with this type of tumor is still copied by one compiler from his predecessor. One still reads of the use of the acetic acid reaction for mucin to make the diagnosis sure.

ETIOLOGY

Adami,[1] in his textbook, describes the nasal polyp as a myxoma, fibroma, adenoma, etc., due to "hyperplastic growth of the mucous membrane of the nose or of the accessory cavities, or to actual tumor formation." Ewing [2] designates it as a fibroma of the nares, nasal polyp, etc., and quotes Chiari's [3] hypothesis of its inflammatory origin.

Zarniko [4] classifies the nasal polypi as: fibroma edematodes simplex; adenofibroma edematodes; fibroma edematodes cysticus.

Up to the time of Billroth there were two clashing opinions regarding the malignancy of the nasal polyp. The one, supporting the malignant theory, based its arguments on the continued recurrence of the polypi after frequent removal. The other school, denying malignancy, based its views on the fact that this type of tumor never formed either local or general metastases.

Billroth was the first to enunciate the theory that the nasal polyp was nothing more than the normal mucous membrane of the nose, which, when stimulated by some irritant or trauma, becomes edematous. He pointed out that the nasal polyp has the same histologic structure as the normal mucous membrane of the nose, that is, columnar epithelium, basal membrane, connective tissue framework and mucous glands (Fig. 2, *C, E, F* and *J;* also Fig. 7). (The comparatively tightly packed connective tissue fibers of the normal mucosa at *J,* Figure 2, become waterlogged and separated from one another in the body of the polyp at *G.*)

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* From the Departments of Oto-Laryngology and Pathology of the Montreal General Hospital.

* Read before the Montreal Medical-Chirurgical Society, March 6, 1925.

1. Adami: Principles of Pathology, Philadelphia, Lea & Febiger.
2. Ewing, J.: Neoplastic Diseases, Philadelphia, W. B. Saunders Company, 1919, pp. 162-164.
3. Chiari: Krankheiten der Nase, 1902, p. 195.
4. Zarniko: Krankheiten der Nase, Berlin, 1910, p. 476.

As pointed out by Hajek,[5] the second great step forward in the development of the true pathogenesis of the nasal polyp we owe to Zuckerkandl.[6] He was the first to demonstrate that the mucous membrane of the middle and superior meatuses of the nose is different from the rest of the mucous membrane of that organ. He showed that the mucous membrane about the ostia of the accessory sinuses and its extension into these cavities was particularly thin, and the underlying connective tissue stroma was exceedingly delicate and loose (Fig. 7 *C*).

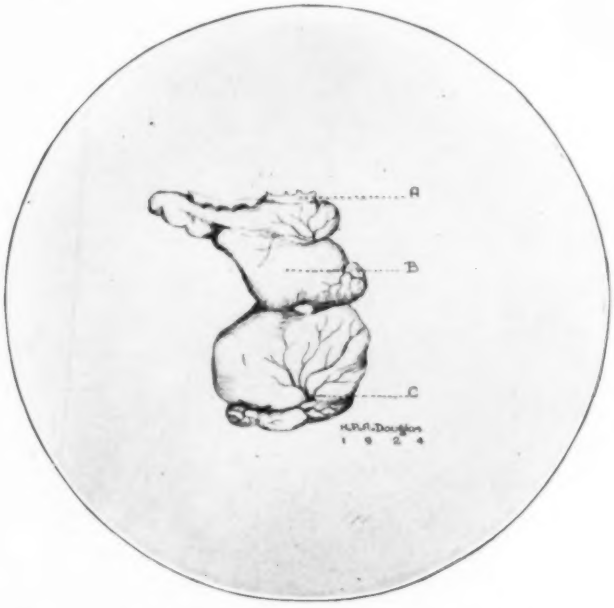

Fig. 1.—Natural sized drawing of polyp. *A*, serrated, honeycombed, bony part of ethmoid dragged away with rest of polyp; *B*, pedicle of polyp; *C*, dilated vein in shrunken body of polyp.

The mucous membrane in these situations is very sensitive and reacts to any severe trauma or irritation by becoming edematous. As a result, it becomes greatly increased in volume. When this succulent mucous membrane happens to be in the neighborhood of, or protrudes through, any of the before-mentioned ostia, gravity causes a constriction at the sharp margin of the ostium through which it protrudes. We now have a pedunculated nasal polyp. As the pedicle passes through the ostium, the weight of the polyp causes an acute bend as it passes over the sharp

5. Hajek: Nebenhohlen der Nase; ibid., Warum recediviren Nasenpolypen? Wien. med. Presse. No. 10, 1902; ibid., Monatschr. f. Ohrenh. **58**, (April) 1924.

6. Zuckerkandl: Normale u. Path. Anat. d. Nasenhohle, Wien and Leipzig, Wilhelm Braunmüller, 1892.

margin of the ostium. This presses against and partially obliterates the lumen of the veins of the polyp at this point; causing a stasis and a further edema of the pendulous mass, which now seeks and occupies the roomiest spaces of the nares. For the third step forward toward the elucidation of this problem, we are indebted to Hajek.[5] He drew attention to the fact that in order to understand properly the pathology of the

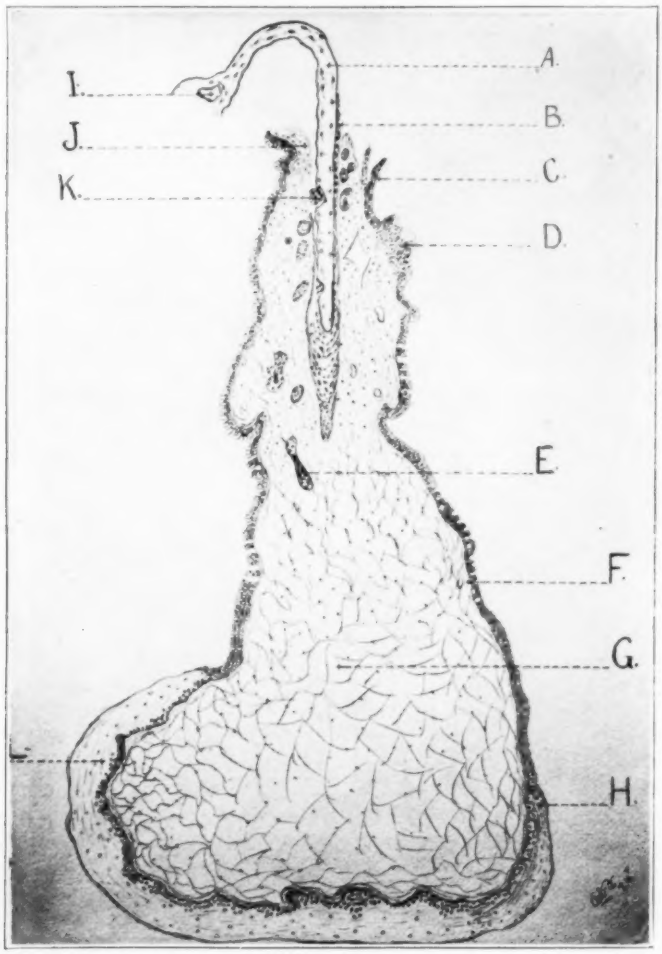

Fig. 2.—Photograph of a drawing of a longitudinal section of the polyp. *A*, part of the ethmoid bone from which the polyp is suspended; *B*, thickened periosteum with row of osteoblasts; *C*, columnar epthelium; *D*, superficial ulcer which has broken through the covering epithelium; *E*, mucous gland; *F*, basal membrane; *G*, edematous stroma of polyp (note the large size of the spaces, the paucity of cells of any description and the entire absence of mucous glands); *H*, borderline between columnar and squamous epithelium; *I*, small ethmoidal cell; *J*, thickness of normal stroma about bone; *K*, osteoclast actively destroying bone; *L*, squamous epithelium.

nasal polyp, one must take into consideration not only the normal mucous membrane of the nose, but also the underlying stroma and the normal bony structure underneath. He pointed out that the loose areolar tissue of the mucoperiosteal layer of the normal accessory sinuses is in close connection with the hollow marrow spaces of the underlying bone (Fig. 8, *a*). The fourth phase of this problem, that the nasal polyp is a gross ocular manifestation of an underlying chronic osteomyelitis of one or more of the accessory sinuses of the nose, I will endeavor to prove.

The subsequent observations are based on the detailed studies of a nasal polyp which I removed from an adult, a woman, aged 45. She

Fig. 3.—Photomicrograph of segment of polyp in region between *D* and *E*, Figure 2. *A*, normal columnar epithelium, *b*, subepithelial abscess; *c*, connective tissue stroma; *d*, mucous glands.

suffered from nasal obstruction and purulent rhinitis for two years. The polyp filled up the vestibule and protruded through the nares to the outside. For the sake of conciseness I leave out the detailed account of the clinical, as well as of the therapeutic, aspect of the case, except that I removed the polyp with a cold snare, as a preliminary measure.

I engaged the loop of the instrument as high as possible, near the attachment of the pedicle to the anterior part of the hiatus semilunaris in the middle meatus, just posterior to the agger nasi. With a sudden jerk, the whole specimen (Fig. 1) came away.

The object which attracted my attention, and which suggested a careful histologic study of the specimen, was the honeycombed piece of bone with the serrated margin which came away with the polyp (Fig. 1, *A*).

Behind this large single polyp were many smaller polypi. These were removed in the usual fashion and nothing unusual was noticed about them. In passing, it may be of interest to note that in every case in which one sees a large obstructing polyp in the anterior nares, this is only a "sentinel" polyp. There are more behind.

Fig. 4.—Photomicrograph of section of polyp in region L, Figure 2. *a*, squamous epithelium; *b*, loose areolar stroma.

Figure 1 is a pen sketch of the polyp, life size. It shows the serrated, honeycombed piece of bone at *A*, from which the polyp springs. This was torn off by the sudden jerk of the snare from the rest of the ethmoid capsule. This sketch also shows the marked dilatation of the veins at *B*.

Figure 2 is a picture of a drawing of a histologic, longitudinal section through the whole tumor; this is suspended from its bony attachment *A* like a pear from its stalk.

At the levels *J* and *C*, the mucous membrane presents the same characteristics as the normal mucous membrane of any part of the nose, that is, it is about the same thickness, has a single layer of columnar ciliated

epithelium, and a thin layer of areolar stroma adjacent to the bone, *J*. At these levels the mucous membrane has been torn from the nose, leaving the projecting piece of bone, *A*, devoid of mucous membrane. At *I*, there is a small ethmoidal cell with its lining membrane. The basal membrane of the columnar epithelium is shown at *F*.

At *G*, one sees the loose connective tissue stroma which contains so very few cells and which is the same type of tissue as at *J*, except that has become water-logged. This forms the bulk of the polyp.

Fig. 5.—Photomicrograph to demonstrate the bony changes: *A*, bone; *ob*, osteoblasts; *oc*, osteoclasts; *d*, mucous glands; *e*, leukocytic infiltration.

Note the comparative absence of mucous glands *E* in the pendulous portions of the tumor. The mucous glands, as may be seen in the drawing, are near the origin of the mass from the bone *A*. Furthermore, it may here be mentioned that this is characteristic of all the nasal polypi that I have examined. There are few mucous glands in the bulk of the tumor; not any more than in the normal mucous membrane. Secondly, wherever they exist they present a normal appearance, and are not tremendously dilated with mucin so as to form the bulk of the tumor and justify the name of myxoma.

That part of the polyp which presented itself to the ouside air and was subject to irritation and friction became covered with a many

layered squamous epithelium, *L* (see also Fig. 4, *A*). This is a beautiful example of metaplasia.

At *H*, is the borderline, in which the columnar cells become changed into squamous epithelium.

At *D* there is a small superficial ulcer, with evidences of a recent inflammation. This shows how the invading infection has broken through the first line of defense, the columnar epithelium.

Figure 3 is a photomicrograph of a segment of the polyp between *D* and *E*, Figure 2. At *b*, Figure 3, one sees where the invading organ-

Fig. 6.—Higher magnification of Figure 5, to show an actual osteoclast at work. *oc*, osteoclast; *ob*, osteoblasts; *A*, bone; *d*, mucous glands.

isms have advanced deeper into the tissues, and have formed a localized abscess. In this section, *a* is the columnar ciliated epithelium; *c* is the stroma, and *d* is the normal mucous glands.

Figures 5 and 6 are photomicrographs to demonstrate chiefly the changes that have taken place in the centrally placed segment of bone, *A*. These photographs were taken to include the "insertion" of the bony part of the ethmoid (Fig. 1, *A*) from the level *C*, Figure 2, to its distal end.

At *OC*, Figures 5 and 6, are the points at which the bone is being actively attacked by infection. There is actual bone destruction going

on as is evidenced by the punched-out margin of the bone. In Figure 6 one can see the osteoclast *OC* actually eating away the bone.

On the opposite margin of the bone, there is an attempt made by nature at new bone apposition, as is evidenced by the piling up of the row of osteoblasts (*OB*), Figures 5 and 6 and *B*, Figure 1.

The bone building osteoblasts cannot keep pace with the bone destroying osteoclasts, as is evidenced by the diseased, irregularly punched-out appearance of the bone; the diseased appearance of the actual bone cells, and, what cannot be shown in an uncolored photomicrograph, situations in which the old bone has been destroyed and the

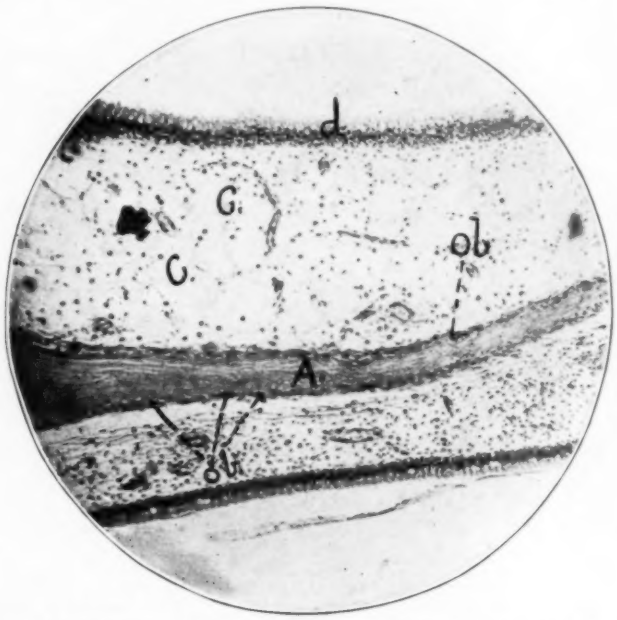

Fig. 7.—Photomicrograph of section of normal ethmoid from child, aged 7 years. *A*, bone (note the regularity of the margins of the bone, as compared with Figures 5 and 6). The thick layer of active osteoblasts; *ob*, on both margins, active growth is taking place in this young bone; *c*, loose delicate areolar stroma the same as in polyp; *d*, columnar epithelium.

new bony matrix has not matured enough to accumulate a calcium deposit. These phenomena are judged by the difference in staining affinities of old bone and new bone for the alum hematoxylin stain.

All the before-mentioned bony changes are pathognomonic of an ostitis or an osteomyelitis, which is the pathologic condition of the piece of included bone in our specimen.

There is a marked leukocytic infiltration of the stroma surrounding the bone in Figures 5 and 6, as compared with the scant cellular elements in the body of the polyp Figure 2, *G*.

CONCLUSIONS

1. The nasal polyp is a localized edema and prolapse of the normal mucous membrane of the nose.

2. This edema may be caused by an infection or by trauma.

3. The great majority, if not all, of nasal polypi are in the neighborhood of, or protrude through, the ostia of the accessory sinuses; that is, their pedicles can always be traced to the middle or upper meatus of the nose.

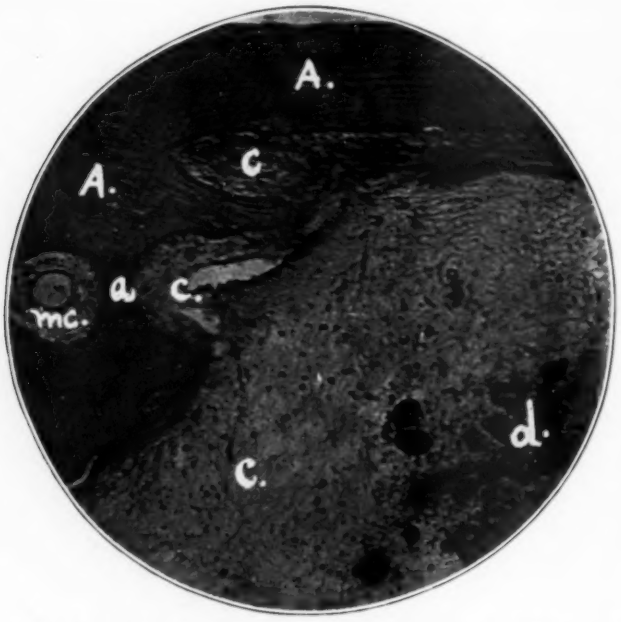

Fig. 8.—Photomicrograph of ethmoid cell of normal child, aged 7 years. *A*, bone; *mc.*, medullary cavity; *a*, communication between areolar stroma; *c*, with medullary cavity; *d*, mucous glands.

4. Because of their situation about the sharp margin of the ostium, gravity causes a compression of the returning vein; this causes a further edema of the body of the polyp.

5. A "full blown" polyp, when not due to postoperative trauma, is always a symptom of bone involvement of one or more of the accessory sinuses of the nose.

6. Free drainage and ventilation of the involved sinus is now made impossible. Because of this stagnation, the infection goes deeper, and results in, first, a periostitis; then an ostitis, and finally an osteomyelitis.

7. Because of the peculiar normal bone histology of the ethmoid (marrow spaces communicating directly with the loose areolar muco-

periosteal stroma, Fig. 8, *a*) and osteomyelitis is easily caused here. Involvement of the bony labyrinth of the ethmoid, therefore, is fairly common when nasal polypi are present.

8. The clinical significance of the last three conclusions is that the nasal polyp is only a symptom of an underlying osteomyelitis, or at least a periostitis of one of the sinuses, depending on the sinus involved. Snaring off the polypi alone will not cure the condition. It will ease the nasal breathing for a while; but the polypi will surely recur, because of the underlying condition. This procedure may be compared to the snipping off of exuberant granulations around the margins of a fistula, due to a chronic osteomyelitis of one of the long bones that needs radical surgical measures. If the ethmoid is involved, the condition is likely to be an osteomyelitis, and all the cells of the ethmoid capsule will have to be cleaned out and removed. If the maxillary, sphenoid or frontal sinuses be involved, the operation of choice ought to be to get a clear view of the involved sinus and to curet out or, better still, dissect off as much as possible of the lining membrane of the involved cavity. If free drainage into the nose is created, the condition will have a chance to clear up.

A CLINICAL STUDY OF BONE CONDUCTION AFTER THE METHOD OF RUNGE *

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Most otologists believe that there is a standard, or normal bone conduction; that is to say, that any certain tuning fork should be heard when its stem is pressed against any part of the head for a length of time, which, for clinical purposes, is constant enough to make it possible to fix a standard for any particular fork that, within reasonable limits, will not vary greatly in any number of normal persons. On this assumption, many tests of hearing by bone conduction have become established, several of which have been universally accepted and used for years in all routine examinations of the ear. It has never been definitely determined, however, whether hearing by bone conduction is, as taught by Bezold, simply the perception of those sound waves which, on their way to the labyrinth, have passed through the sound conduction apparatus; or whether, as argued by Wittmaack and others, there is a direct craniolabyrinthine transmission of the sonorous vibrations. The question is an important one, for, under the Bezold theory, all sound waves which reach the labyrinth directly, that is, without appropriate intervention of the sound conduction mechanism, are inaudible; consequently our hearing tests by bone conduction are simply additional methods of diagnosis in lesions of the conduction apparatus and only of suggestive value in determining the presence of perception deafness. On the other hand, if there is, at least in part, a direct craniolabyrinthine transmission, our bone conduction tests take on an added significance, and hearing by bone conduction can be considered as a phenomenon separate and distinct from physiologic air conduction and of greater value in differential diagnosis.

WATER-FILLING TEST

In 1923, H. G. Runge [1] published a detailed study of bone conduction in which he described his "water-filling" test and the results obtained therewith. The test is made, first, by taking the patient's bone conduction with the fork on the mastoid; the head is then placed over to one side, the ear filled with 1 c.c. of water and the bone conduction again taken. Runge found in the normal ear that "water-filling" about doubled the duration of perception by bone conduction up to C^2 fork

* Read before the American Otological Society, Washington, D. C., May 5, 1925.

1. Runge, H. G.: Ztscher. f. Hals-, Nasen- u. Ohrenh. **5**:289, 1923.

(512 double vibrations); above this pitch the reaction was not so pronounced. He further found that in pathologic cases the results were entirely different.

1. After the radical mastoid operation, "water-filling" did not increase the bone conduction.

2. If there was evidence of complete fixation of the stapes, the test did not increase the bone conduction.

3. If there was only partial ankylosis somewhere in the ossicular chain, the "water-filling" gave only a moderate increase in the duration of bone conduction.

4. If there was evidence of cochlear degeneration, bone conduction which was greatly reduced could usually be somewhat increased by the "water-filling."

5. In acute otitis media, bone conduction could be slightly diminished or at least not increased by the test.

The test interested me because it is easy to carry out, without discomfort to the patient and apparently offered a means of obtaining useful clinical information. I, therefore, began to employ it rather extensively in routine work, and in a variety of conditions, namely, (1) chronic progressive deafness of various middle-ear types; (2) acute purulent otitis media; (3) chronic purulent otitis media; (4) after the radical mastoid operation, and (5) perception deafness.

FINDINGS

The findings in a series of more than fifty patients corresponded almost exactly with those quoted from Runge; and from a practical standpoint the test appeared of especially valuable diagnostic significance in those conditions for which we seem to have no better term than the chief symptom, chronic progressive deafness. Failure to increase bone conduction by the Runge test has occurred most frequently in those patients in whom increased bone conduction already exists, and who present other signs of stapedial fixation or of profound disturbances in the conduction mechanism. In these patients one cannot expect treatment to be of benefit. On the other hand, if bone conduction could be increased, some gain in hearing has usually been obtained.

My interest in the test further increased when I realized that with it one can easily reproduce and control for study in the normal ear, various abnormalities of bone conduction which occur in pathologic conditions giving rise to deafness, such as a definitely referred Weber test, an increased Schwabach and variations of the Rinné tests. Variations of these reactions as they occur in deafened patients could also be studied from a somewhat different angle.

In my experiments with the method I have made routine use of a C^2 fork (512 double vibrations), which I have used in bone conduction tests for at least ten years. I feel, after studying hundreds of tests of my own bone conduction and others, that the fork should be normally heard when placed over the mastoid for not less than 20 seconds nor more than 25 seconds. For clinical purposes, however, nothing over 15 seconds is considered as reduced bone conduction and nothing less than 30 seconds is considered as increased. It is set into vibration by being allowed to fall by its own weight on my knee. I have selected the fork of this pitch because I feel that it must be heard and not felt. In normal persons, bone conduction taken over the mastoid with this fork can be doubled by filling the ear with 1 c.c. of water.

One of my first observations in the use of the Runge test was to the effect that if the labyrinth is normal, it is only possible to obtain a marked increase in bone conduction if a completely mobile ossicular chain exists. No increase in bone conduction could be elicited in several patients who had had the radical mastoid operation and who have no evidence of labyrinth lesions; no increase could be obtained in patients of the otosclerotic type, in whom bone conduction already showed a marked increase before the "water-filling," and no increase, but rather a slight decrease, occurred in acute suppurative otitis media and particularly in chronic suppurative otitis media. On the other hand, several patients with possible labyrinthine lesions and marked decrease in bone conduction showed a striking gain in bone conduction when the ear was filled with water.

REPORT OF CASES

CASE 1.—D. S., a man, aged 73, complained that he missed the notes of the violin at concerts, certain words and certain voices. Examination showed perfectly normal drumheads. Hearing tests showed a rather negligible loss in the low scale, but from C^2 (512 double vibrations) to C^5 (4,096 double vibrations), there was a definite falling off in duration of perception. The left ear was slightly deafer than the right. The bone conduction over the right mastoid was 10 seconds; over the left, 7. "Water-filling" brought the bone conduction up to 17 seconds on the right side, and to 20 seconds on the left.

CASE 2.—T. D., a youth, aged 19, has been under my care at intervals for four years. At the first examination, on Aug. 23, 1921, he had all the evidences of a perception deafness in both ears; negative bone conduction, with marked loss for the high notes and reduced vestibular responses. The origin of his deafness seemed to be toxic as it followed severe tonsillitis. Under appropriate treatment he showed marked improvement, although the deafness recurs at intervals, usually as a result of a nasal or throat infection. It, however, never reaches the same degree as when the patient was first seen by me, and is always more marked in the right ear than in the left.

The case is typical of those reported by Francis P. Emerson, in support of his theory of the toxic origin and the perception defect in so many patients. The bone conduction improves with the hearing, and has been as good as 15 seconds in the right ear and 12 seconds in the left; it always becomes negative with the recurrence of deafness. During February, 1925, his hearing

became slightly affected following an acute rhinitis. His bone conduction was negative in both ears. "Water-filling" brought out a Weber referred to the right ear and bone conduction for 26 seconds over the right mastoid. Water in the left ear referred the Weber to the left and gave a bone conduction over the left mastoid of 15 seconds.

The following history (CASE 3) is in striking contrast to the preceding case and somewhat indicates the value of the Runge test in differential diagnosis.

CASE 3.—F. D., a woman, aged 20, when a child, aged 8, was under my care in the Dispensary at the Baltimore Eye, Ear and Throat Hospital. She was becoming very deaf at that time, without apparent cause, though all efforts were made to stay the process. There was no family history of deafness. The Wassermann test was negative and she presented no evidence of congenital syphilis. The tonsils and adenoids were removed, and as far as possible her general health was looked after with more than usual care. No improvement was perceived, and finally the patient left the clinic. She came to my office in January, 1925, almost totally deaf in the right ear and hearing with the left only words spoken loudly at a distance of 3 feet. As was to be expected, all the evidences of labyrinthine degeneration were present, with no hearing by bone conduction in either ear, and greatly reduced vestibular reactions. Her ear drums were slightly atrophic in appearance but the handle of the malleous moved freely on massage in both ears. "Water-filling" had no effect on the right ear, but gave 9 seconds of perception over the left mastoid.

COMMENT

It was quite evident in both Cases 2 and 3 that the conduction apparatus was properly functioning. The surprising increase in bone conduction brought out by "water-filling" test in Case 2 would certainly indicate that there was little or no disease of the nerve elements within the perception apparatus. In Case 3, every clinical sign pointed to a degeneration of the neuro-epithelium within the labyrinth. In Case 2, the patient obtained marked relief from treatment, which consisted primarily in the clearing up of the nasal or throat infection and from the use of inflation by means of the eustachian catheter. In view of the facts that his ears have never shown any marked evidence of negative pressure, that the tympanic tubes are not stenosed and are easy to catheterize, that in conjunction with the other evidences of perception deafness already given there is always a definite falling off in duration of perception for the C^5 fork (4,096 double vibration), the use of the catheter would seem rather illogical, if not distinctly contraindicated. It is not unusual, however, to find patients who are helped by inflation, in whom its employment seems entirely unnecessary. Kerrison has brought out this point in his textbook and gives the report of one very striking history to prove it. It is the hope, perhaps, that a patient's deafness may be of this type that makes the use of inflation almost a fetish.

The histories of these two patients have been contrasted and gone into at length, to bring out the value of the Runge test in apparently making it possible to determine the difference between what may be a perversion of the conduction function within the labyrinth and a disease

of the true perception elements. The conveyance of vibration through the cochlea, with the resultant sympathetic resonance within the duct of the cochlea, is quite as important as the conversion of these impulses by the organ of Corti into nerve stimuli; it is certainly possible that disturbance of the normal relationship of those labyrinthine structures whose functions are physical could give rise to a type of deafness in which the clinical signs and tests would point to a lesion within the internal ear indistinguishable from a degeneration of the neuro-epithelium. It seems possible to make such a differentiation with the Runge experiment so long as the ossicular chain is mobile. The ability to increase bone conduction markedly demonstrates, in my opinion, the mobility of the middle ear structures, and certainly proves that the nerve endings are healthy enough to be stimulated. The use of a single tuning fork of one pitch is not always sufficient, and interesting observations can be obtained in so-called partial perception deafness by the use in the Runge test of several forks. In one of my patients showing negative bone conduction for the C^2 fork (512 double vibration) with marked reduction in duration of perception for the C (128 double vibration) and C^1 forks (256 double vibration), the Runge test did not bring out any perception for the C^2 fork (512 double vibration) but increased the duration of the C^1 (256 double vibration) from 6 seconds to 12 seconds and the C (128 double vibration) from 11 to 20 seconds. This test apparently confirmed the findings of the test by air conduction, which had shown a loss through the whole scale from C_2 (64 double vibration) to C^5 (4,096 double vibration), but much more marked from C^2 (512 double vibration) up.

If there is evidence of marked and extensive involvement of the organ of Corti with negative bone conduction, the Runge test is of course negative for all forks.

THE RUNGE TEST

Early in the routine use of the Runge test, I realized that the "water-filling," if positive in its action, should produce a definite Weber reaction. This was easily proved on a normal person and in those patients with normal or reduced bone conduction that could be increased even moderately. My first impression—that if the Runge test is to produce increased bone conduction it is necessary for the ossicular chain to be freely movable—was further strengthened by the observation that in patients with definitely increased bone conduction, or in those with normal bone conduction which could not be lengthened, the sound of the tuning fork placed on the forehead was not referred to either ear. It next occurred to me that the experiment furnished a way of studying the Schwabach test with tuning fork on other portions of the head, for many eminent otologists make this test from the forehead or vertex;

and Bezold has said that the duration of bone conduction need be ascertained only from the vertex for both ears at the same time, and not from each ear separately from the mastoid process, as it is almost impossible to exclude the other ear. We all of us are familiar with contra-audition, as it was called by the early investigators, in those patients with conduction deafness showing marked difference in the hearing of the two ears, and with a definite Weber reaction which refers bone conduction from all over the head to the deafer ear. This can be beautifully reproduced with "water-filling." The following experiment was performed on a young man, aged 20, who had never had any ear trouble, who could hear a low whisper at 30 feet in each ear, whose drumheads apeared perfect and whose range of hearing was normal. The duration of bone conduction with the C^2 fork (512 double vibration) in the midline of forehead was 22 seconds. The duration of perception over the right mastoid was 26 seconds; over the left mastoid, 25 seconds. The right ear was then filled with 1c.c. of water. The sound of a fork held over the forehead was definitely referred to the right ear and apparently heard in the right ear for 30 seconds; over the right mastoid, it was heard for 45 seconds, and when held over the left mastoid, it was heard for 35 seconds. The left ear was then water-filled, and the sound of a fork held on the forehead was heard in the left ear for 38 seconds; when the fork was held over the left mastoid, the sound was heard for 50 seconds, and when held over the right mastoid it was heard for 37 seconds. The experiment, repeated several times, indicated that in the normal ear, "water-filling" will, apparently, increase bone conduction from any part of the head, but the sound will be referred to the blocked ear so long as the fork is audible and the duration of perception will always be the longest over the mastoid of that ear. Is it not possible, therefore, to formulate the rule *that when bone conduction is increased by the Runge test the final tone of the tuning fork is entirely perceived by that ear which is water-filled?* This rule seems to hold good in all pathologic cases in which the "water-filling" produces an increased bone conduction with a definite reference of sound to the obstructed ear; therefore, I changed the usual way of making the test and adopted the following routine: (1) the Weber test from the forehead; (2) the duration of perception over each mastoid; (3) the right ear was filled with water, the Weber again taken from the forehead; (4) the bone conduction was taken over the right mastoid; (5) the bone conduction was taken over the left mastoid, the right ear remaining blocked; (6) the left ear was filled with water and the tests repeated.

Testing in this manner has given interesting results.

Mrs. J. DeR. heard with the right ear words spoken at a distance of 3 feet; with the left ear, words spoken at a distance of 2 feet. The Weber test was

referred to the right; the right bone conduction was 27 seconds; the left bone conduction was negative.

The Runge Test: The right ear was filled with water and the Weber test referred to it. The right bone conduction was 22 seconds (note slight decrease); the left bone conduction, 7 seconds referred to right. The Runge test was then applied to the left ear: the left bone conduction was 12 seconds, and the right 23 seconds.

An analysis of these findings will indicate that all hearing by bone conduction is from the right ear.

THE "WATER-FILLING" TEST

My clinical findings with the "water-filling" test have so nearly duplicated those given by Runge that I have presented them simply in confirmation of his pioneer work, and with no claim of originality for any slight variations in the methods and observations. I do not, however, agree with his explanation concerning the increase in the duration of bone conduction brought about by the "water-filling." He believes that increased bone conduction is occasioned by the improvement in conduction produced by the water on the drumhead, partly by a diminished "outflow" or escape of sound from the ear, the former being the chief factor and the latter a supplementary influence. It is easy to prove that filling the canal with water does not improve the conduction of sound waves to the ear-drum but that, as a matter of fact, it reduces the hearing by air conduction very considerably, even when the stem of the tuning fork is placed in the fluid so that the vibrations are directly transferred to the drumhead. I have proved this to my own satisfaction in several normal persons, in patients with chronic progressive deafness of otosclerotic type, and in patients who have had radical mastoid operations. I, therefore, cannot understand this portion of Runge's explanation, particularly as it is controverted by the experiment itself in those patients in whom it produces no increase or rather a decrease in bone conduction. The "outflow" theory of Mach has also never been proved and in some respects is physically untenable.

THE BEZOLD TEST

It is obvious that the reaction obtained in a normal ear by "water-filling" is a near duplication of the Bezold triad symptom complex, for one must admit that a column of water weighing approximately 1 gm. resting on the drumhead must produce a fixation of the ossicular chain simulating the stapedial ankylosis of otosclerosis. But the Bezold hypothesis that increased bone conduction is brought about by the vibrations striking the edge of the drumhead and annular ligament and not the flat surface of the drum, and that increase in tension anywhere in the ossicular chain or its connections reduces hearing by air but increases it by bone, is not confirmed by his own contention that all hearing by bone conduction must be through the tympanic mechanism.

If we accept this theory, we must assume that the stapes alone, as after the radical mastoid operation with increased bone conduction, or a fixed bar in otosclerosis, rather than a system of highly specialized levers, is all that is necessary in either case to perform what we accept as the function of the drumhead and ossicular chain, namely, the transforming of motion of great amplitude and little force into a motion of small amplitude and great force best suited to be transferred to the labyrinthine fluids, or, according to Bezold, to convert longitudinal into transverse vibrations. The Bezold theory would fully explain the Runge reaction, if it were not for these paradoxical elements.

SYMPATHETIC RESONANCE

It is more than probable that hearing is dependent on sympathetic resonance at some point in the labyrinth. This, I think, has been granted in all theories of hearing accepting a peripheral tone analysis, from Helmholz down. Induced or sympathetic vibrations are always of less intensity than those of the exciting force on account of a leakage of energy; therefore, when the stem of a tuning fork is placed on the bone of the head, is it not possible that we hear it by the direct creation of sympathetic resonance in that portion of the internal ear tuned to respond to the pitch of the fork used? The duration of perception is much less than by air conduction, under the law of induced vibration, even though it may be augmented in the normal ear by vibrations taken up by the conduction mechanism. If this supposition is admitted for argument only, then bone conduction may be considered as an entity, separate and distinct from physiologic air conduction, and its abnormalities may be explained as occurring not because of faulty or anomalous transmission of sound waves by the drumhead and ossicular chain, but as due to faulty or anomalous perception brought about by changes in the position of intralabyrinthine structures which must occur during forced and abnormal inward movements of the foot plate of the stapes, and the compensation movements outward of the membrane in the round window or when fixation of the stapes results in a motionless fluid within the internal ear.

The Runge experiment seems to indicate the possibility of this explanation for the following reasons:

1. To obtain a marked increase in the duration of bone conduction, it is necessary that the movements of the ossicular chain be perfectly normal.

2. No increase in the duration of bone conduction can be obtained in those persons in whom the drumhead and ossicles have been removed, or in those patients presenting evidence of complete fixation of the ossicular chain and showing either increased or normal bone conduction.

3. The amount of increase in the duration of bone conduction over the normal seems dependent on the degree of mobility of the middle ear mechanism, for in partial fixation only moderate increase in the duration of perception over the normal can be obtained.

4. Bone conduction can be doubled in duration of perception in normal and in deafened persons so long as the ossicular chain is freely movable.

5. Hearing by bone can often be elicited in patients in whom the bone conduction is negative before the "water-filling."

6. In certain persons presenting negative bone conduction for a fork of one pitch and a positive bone conduction for other forks, the fork which is not heard will remain negative on "water-filling," but the other forks may become definitely and normally increased.

CONCLUSIONS

This report is simply a repetition of the investigations of Runge. I have applied the test in everyday clinical work; I have found it of definite and distinct value in the diagnosis of complete and partial stapedial fixation, and in differentiating between conduction and perception deafness. The experiment can be repeated in the physical laboratory with definitely controlled air pressure and I believe that it can be shown that bone conduction can be increased up to a certain maximum in direct proportion to the amount of pressure employed. The Bezold theory concerning increased bone conduction has never been fully acceptable; direct craniolabyrinthine transmission of sound waves based on sympathetic resonance is not impossible, and changes in the tension of intralabyrinthine structures must take place. These are many points which can be solved only by the physicist interested in otology and the otologist interested in physics working together, for otherwise the physicist sees only the normal, and the otologist, as Gradinego has well expressed it, "sees only a deformed likeness of hearing." As a clinical otologist, I feel that hearing by bone conduction may be, for diagnostic purposes at least, considered a distinct entity, but I am ready to be otherwise convinced.

THE NASAL (SPHENOPALATINE) GANGLION

IN RELATION TO ITCHING IN THE AUDITORY CANAL AND TO TINNITUS AURIUM

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ITCHING IN THE EARS RELIEVED BY NASAL GANGLION TREATMENT

In the February, 1925, issue of the *Laryngoscope,* I recorded an interesting case of persistent itching in the external auditory canal, relieved by nasal ganglion treatment:

CASE 1.—A physician, one of my students at the New York Post Graduate Hospital, Dr. A. B. L., of Kingston, Ont. For the last six years he had suffered from severe attacks of itching in the ears, especially when using a stethoscope. The annoyance was so great as to induce him to study otolaryngology, to avoid constant use of the stethoscope. The itching would be so severe as to awaken him sometimes at night and the scratching lead to repeated formation of furuncles. There were no typical headaches or neuralgias. Local treatment of all sorts was of no avail.

Nasal examination revealed a chronic bilateral ethmoiditis with polypi in both middle meati. He had chronic tonsillitis.

Local application of saturated solution of cocain to both ganglions gave immediate relief. He received two treatments and has since then been free from any itching in the canals.

The following are additional cases:

CASE 2.—*Tinnitus Aurium.*—A woman, R. G., aged 22, was referred to me by Dr. H. Handelman. She complained of noises in the head and of persistent itching in both ears for the last year, during which time she suffered from frequent atttacks of sneezing and rhinorrhea. She had good nasal breathing, normal hearing and no headaches. She observed that occasionally when the head noises were bad she had a feeling of soreness in the throat.

Examination revealed the left middle turbinate hypertrophied, the mucous membrane thick and soft and the right middle turbinate apparently normal. Posterior rhinoscopy revealed a bilateral posterior ethmoiditis with moderate amount of mucopus. The patient had small buried tonsils and carious teeth. Both extermal auditory canals were scratched, the left was swollen and eczematous.

Local application to the nasal ganglions gave immediate relief from both the noises and the itching. She had a mild recurrence two days later, and fortunately came in when Dr. Greenfield Sluder was visiting at my office. I had the pleasure of demonstrating the case to him. Dr. Sluder cocainized one side and I cocainized the other. The patient was immediately relieved and has remained free from the noises and itching since the end of April. Examination on May 19 showed both external canals healed and free from scratch marks. There has been no recurrence.

CASE 3.—J. B., a woman, came to me during an attack of furunculosis of the auditory canal, complaining of intense pain and itching. She received considerable relief from nasal ganglion applications. Nasal examination showed intense redness in both ganglion regions. She had chronic tonsillitis and carious teeth. I performed a tonsillectomy after the furuncle had healed, had the carious teeth treated and gave her nasal ganglion applications. She has had one attack of furunculosis since the onset of treatemnt but has been free from itching.

I was first led to investigate the relationship between itching of the ears and the nasal ganglion by observing the frequency of the complaint of itching in the eyes, nose, throat and ears associated with the typical nasal ganglion neuralgias. Subsequently, I observed that the symptom of itching ears can appear without any other indication of nasal ganglion disturbance.

CASE 4.—This is a case of typical nasal ganglion neuralgia associated with itching of the canals. Mrs. A. J., aged 43, was referred to me by Dr. J. Miller, on May 20. She complained of headache extending from the back of the left eye into the left temporal region, left mastoid and down along the sternocleidomastoid muscle. During attacks she became nauseated, had itching in both ears and severe cough, enough to awaken her at night. During the four days prior to consulting me, she developed severe hoarseness. She also suffered from considerable deafness of both ears.

Nasal examination revealed both posterior tips of the middle turbinate bluish and covered with a small amount of thin yellowish discharge. She had chronic tonsillitis with enlargement, and acute laryngitis.

Cocainization of both nasal ganglions relieved the itching immediately. She has had four applications to both ganglions and has remained entirely free from neuralgia since her first visit. The itching has recurred to a mild degree but can be relieved immediately by a local application of cocain to the nasal ganglion.

The itching sensation in the eyes is often interpreted as a sandy feeling usually associated with a heaviness of the eyelid. Such patients receive all sorts of local treatment of the eyes with little or no relief.

CASE 5.—Mrs. I. S., aged 53, for the last six years had been complaining of a sandy feeling in both eyes associated with a heaviness of the lids, making it difficult for her to keep her eyes open. She also complained of a sensation of dryness in the nose. There were no other complaints. Examination revealed an atrophic rhinitis, deflected septum with a spine impacting and obstructing the left middle meatus, chronic tonsillitis and carious teeth.

Local application to both ganglions gave immediate relief. Since the first application the recurrences have been very mild and the periods of relief much greater than previously.

COMMENT

These patients were particularly fortunate in being so readily relieved. There are, as is well known, other conditions that are stubborn and require alcoholic injection of the ganglion.

In the February, 1925, issue of the *Laryngoscope*, I gave a detailed description of the anatomy of the nasal ganglion indicating its wide connections and describing the probable route of the impulses. I advocate injection of the ganglion by way of the palatomaxillary canal.

351 West Eighty-Sixth Street.

PROGRESS IN OTOLARYNGOLOGY

A Summary of the Bibliographic Material Available in the Field of Otolaryngology

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ACUTE AND CHRONIC SUPPURATIVE OTITIS MEDIA

INCLUDING ACUTE INFECTION OF THE BLOOD STREAM

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For the purposes of a critical résumé, recent additions to the literature on otolaryngology may conveniently be divided into three classes, each requiring a different method of treatment. The first includes case reports and theses which are corroborative of previously accepted work. While such papers enrich our sources of reference, to discuss their details in a summary of this kind would take us too far afield. The second group comprises articles which mark a definite and recognizable advance. The salient points of these must be discussed in the light of our present knowledge. The third and last division embraces those contributions which present new ideas that cannot be immediately accepted, but which must be discussed and studied before a final judgment of them is made. As far as possible, all the material commented on in all the groups is such as would tend to stimulate scientific discussion.

ETIOLOGY FACTORS IN THE OTITIDES

In regard to the incidence and predisposing causes of otitis media, Smith [1] has made some interesting observations in a study of 613 consecutive hospital admissions among children. Of this number, 33.4 per cent. suffered from otitis media which was either present before, or developed in the course of the hospital stay. Most of these children were artificially fed. There was no relationship to be noted between race or sex and the incidence of the otitis. The influence of seasonal variations was marked, with the greatest number of cases occurring during the winter months. Age likewise played a part: 50 per cent. of the cases with ear involvement were between 3 and 15 months of age, while 7.4 per cent. were between the ages of 5 and 10 years. The age of the patient had no bearing, however, on the duration of the disease. The use of various means to prevent the spread of infection by contact and droplet did not reduce the number of cases of otitis media which

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1. Smith, D. T.: Causes and Treatment of Otitis Media. Observations on Two Hundred and Eighty-five Cases Occurring in Six Hundred and Thirteen Consecutive Hospital Admissions, Am. J. Dis. Child. **28**:1 (July) 1924.

developed during hospital confinement. The type of disease which was the forerunner of the otitis had a great influence on the occurrence of the ear infection. Thus, 50 per cent. of the otitides developed in patients suffering from some infectious disease, such as pneumonia, dysentery, nasal diphtheria, pertussis or pyelitis, while only 21 per cent. developed in children suffering from noninfectious diseases, nephritis or prematurity. Smith thinks that otitis media in some way may be associated with vitamin deficiency causing a lowering of tissue resistance.

There are many other factors which might be considered in connection with the relationship that the age of the patient and the type of forerunning disease have to the occurrence of otitis media. The observation that the greatest number of cases were noted during the first year of life and in association with the infectious diseases of childhood is in accord with the generally accepted view. The peculiarities of the infant's upper respiratory tract have an etiologic bearing on the occurrence of otitides in childhood more important than that of a disturbed vitamin metabolism. It is true that the latter may tend to predispose the child to disease, per se, and thus more easily lay him open to attack by one of the exanthems. Once the patient has been subjected to an exanthematous infection, however, the factors to which Wittmaack [2] called attention play their rôle in the production of the otitis. The tissues of the middle ear spaces and the bronchotracheal tree are of similar embryonic origin, and even in their postembryonal state they show striking resemblances. It is natural, therefore, that infections selecting the upper respiratory tract should also attack the similar tissue in the middle ear spaces. Finally, the anatomic peculiarity of the eustachian tube in the infant favors the spread of infection, by contiguity, from the nasopharynx to the ear; and it is well known that the nasopharynx is the site of inflammatory reactions during the exanthems.

The presence of ear disease which had been undiscovered during life is discussed by Southby [3] in a study of fifty consecutive necropsies. Thirty-two of these cases were patients under 2 years of age. In thirty-three instances, a gross purulent infection of one or both middle ears was present; yet, in twenty-nine of these both drums were intact. It is remarkable to record that these children had been examined while in the hospital and yet no discoverable redness nor bulging of the membrana tympani had been observed. In fact, there was no symptom noted during life to call attention to the condition of the ears. Of the cases included in this group, there were eleven that showed a purulent meningitis of otitic origin. Among the other seventeen patients, there were six with meningitis which was not of otogenic derivation.

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2. Wittmaack, quoted by Janse, R.: Pathologische Anatomie des Ohres, Leipzig, 1912, p. 48.

3. Southby, R.: Symptomless Otitis Media of Infancy and Childhood. Some Postmortem Observations, M. J. Australia **1**:410 (April 26) 1924.

The occurrence of such middle ear infection without apparent clinical changes in the otoscopic findings is recorded in the older literature. The reason must be sought in the peculiarity of the infant's eustachian tube, which, being short and straight and having an open lumen, permits of easy egress for the pus from the middle ear into the pharynx. Since there is no back pressure of pus in the tympanum, there is no bulging of the drum. Finally, the difficulty in estimating the otoscopic picture in infants is also a contributing cause of the failure to recognize tympanic suppuration intra vitem.

In this last connection, Rendu's [4] recommendation of routine aspiratory puncture of the tympanic membrane in all children in whom there is an infectious syndrome of unknown origin is worthy of discussion. He employs a three-ring syringe furnished with a needle. This is inserted through the drum at an obtuse angle under complete visual control. When pus is present, it is immediately demonstrable and the puncture point can then be transformed into a paracentesis opening. When pus is not present, the danger of carrying infection into the middle ear is small when compared with the exploratory myringotomy. This is a purely relative superiority, however, for it would hardly seem that an exploratory myringotomy is ever indicated at the hands of an experienced aurist. Pus usually demonstrates its presence in the tympanic cavity by its pressure effects on the drum. The procedure advocated by Rendu may find its value in the rare case in which the presence of an otitic purulency is uncertain from the clinical standpoint, but in which there is reason to suspect it because of suppuration at the pharyngeal orifice of the eustachian tube. It should be considered only when diagnosis remains uncertain, despite all the other means at our command.

That the eustachian tube is a factor in such cases has long been a moot point. Brunnetti [5] approaches the problem from another angle. He considers otorrhea of the eustachian tube as a separate pathologic entity, and states that it is due to an inflammatory process of the adenoid tissue in and about the tube. He finds this condition in lymphatic subjects, and more commonly in children than in adults. The lesion may suppurate into the pharynx, which is usual; or, if there is an opening in the drum, the discharge will pass through to the external ear. It seems to us that this added classification of eustachian tube purulency is unnecessary. If there is a suppurative focus which discharges through the middle ear, the case is one of purulent otitis media. The origination of otitides in infections of the nasopharynx is too well understood to

4. Rendu, R.: De la ponction aspiratrice du tympan dans le diagnostic de l'otite du nourrisson, Arch. internat. de laryngol. **30**:274 (March) 1924.

5. Brunnetti, F.: Otorrhea of the Eustachian Tube, Arch. ital. di otol., **35**: 135 (April) 1924.

require further discussion here. Suffice it to state that the condition described by Brunnetti in no way differs from the repeated attacks of purulent rhinitis or otitis which are of frequent occurrence in children and which readily clear up after proper attention to the nasopharynx.

Regarding the presence of nasal suppuration in association with the otitides of childhood, a direct relationship between the two has been established. In a study of forty-two consecutive cases of surgical mastoiditis, Watson-Williams [6] found the nasal accessory sinuses to be infected in twenty-eight instances. In twenty-five of these, the nasal trouble was located on the same side as the aural lesion. Many of these infections were in children. The offending organisms were either the pneumococcus, the streptococcus or the staphylococcus. These observations may throw some light on the etiology of recurrences of mastoiditis after operation. Many of the patients on whom operation is performed again reveal perfectly performed mastoid operations; and no local cause can be found to account for the recurrence. Perhaps more thorough investigation of the nasal accessory sinuses in these cases may reveal the focus of infection. Many of them may possibly be spared repeated attacks of aural suppuration by clearing up the nasal condition.

The influences of heredity on the purulent otitides in children engaged the attention of Lubet-Barbon and La Bérnadie.[7] These observers studied a series of cases in which a perforation of the drumhead existed in the region of the fissure of Rivinus. This condition was most frequently present on both sides, and the authors believe that such patients have a congenital predisposition to suppurations that evacuate themselves through this opening, which is, of course, a locus minoris resistentiae. They also state—and rightly so—that this area of the drum is a natural exit for pus coming from the ossicles or the attic. However, it seems more logical to assume that what these observers noted were epiretrotympanic infections, such as commonly occur in infancy and childhood during the period of susceptibility to the exanthems. It is more usual to see perforations in Shrapnell's membrane in children than in older patients; and certainly bilateral aural lesions are of more frequent occurrence in childhood than in later life. Thus it would seem that the occurrence of bilateral otitides in infancy and early childhood becomes comprehensible on an anatomic basis rather than on the grounds of heredity. The latter, excepting when syphilis is present, is not regarded as a factor in the production of the aural lesions of childhood.

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6. Watson-Williams, P.: The Clinical Importance of Nasal Sinus Infection in Mastoiditis, J. Laryngol. & Otol. **39**:561 (Oct.) 1924.

7. Lubet-Barbon and La Bérnadie: Bilatéralité des lésions de la membrane de Shrapnell, Ann. d. mal. de l'oreille, du larynx **42**:1171 (Dec.) 1923.

PATHOGENIC FACTORS

Valentine [8] studied the bacterial flora in middle ear suppurations, both acute and chronic, with a view to determining the possible elements contributing to chronicity. The bacterial studies in acute cases were made directly following paracentesis and then at intervals during the continuance of the discharge. It was found that in nearly all the cases the beta hemolytic streptococcus was the most important etiologic organism. While this was found soon after paracentesis in pure culture, studies made several days later revealed the presence of other varieties of organisms, with a marked reduction in the number of beta hemolytic streptococci. In some instances a different fermentative group of streptococcus supplanted the original variety of beta hemolytic streptococcus. Valentine suggests that the secondary invaders may be responsible for the chronicity of a lesion.

This paper would have been of much greater value had the types of cases studied been grouped more thoroughly instead of being considered under so inexact a classification as that of acute and chronic middle ear infections. In the type of chronic aural disease in which the lesion is limited to the tympanic mucosa, it has usually been found that the organism which is present has no bearing on either the type or the duration of the lesion. The causative factor in the production of chronicity in these cases is always to be found in the nasopharynx. When pathology there predisposes to repeated aural suppuration by the propagation of infection through the eustachian tube, the type of bacteria found in the aural discharge plays no rôle. On the other hand, in those cases of chronic suppurative otitis media in which chronicity is due to the destruction of bone, there too the type of the bone lesion must be determined. A cholesteatoma or an infiltrating new growth is of itself capable of destroying bone and producing a persistent aural discharge. The bacteria found in such instances are regarded as secondary, concomitant invaders, and not as the causative factors of the lesion. It is in the type of chronic suppuration which is due to bone caries that bacterial etiology plays a part, not yet wholly understood. There are undoubtedly some organisms or strains of certain organisms that exhibit a selective action for bone. Why should the streptococcus strain in scarlet fever or in the more virulent form of streptococcus sore throat predispose toward chronicity of the aural suppuration, with bone necrosis, while other forms of streptococcus in otitic disease produce nothing but an acute infection which runs its course and subsides? With our present knowledge, we are unable to utilize the findings on bacterial examination of otorrhea to classify cases. We agree with

8. Valentine, E.: Bacteriologic Study of Middle Ear Infections, J. Infect. Dis. **35**:177 (Aug.) 1924.

Valentine that it is improbable that a specific microorganism may be considered as a common primary or secondary factor in the production of chronicity.

Bussacca [9] also found the bacterial flora of chronic suppurations to consist of mixed infections. In five of the ten cases examined by him, he found spirochetes associated with fusiform bacilli. He attempted to treat those five cases with various spirocheticidal remedies, but with varying results. Here again the effort to treat chronic suppurations of the aural cavity by attacking the organisms found in the otorrhea is contrary to the pathogenesis of the lesion. Unless the organism found is specific in nature, such as *Spirochaeta pallida,* diphtheria bacillus or tuberculosis bacillus, each of which produces characteristic lesions, treatment instituted to destroy the bacteria will be found futile, in the majority of instances, in halting the advance of the lesion. The bacteria found in aural discharges of long standing are to be regarded as secondary invaders which have found fertile soil in an area previously devitalized by the original causative factor, whatever it may be. For example, streptococci found in a cholesteatomatous discharge would never be regarded as the cause of the chronic otorrhea in such a case.

Coming now to reports of specific organisms in the middle ear, Cemach's [10] studies of tuberculosis of the ear are worthy of note. In his second paper, he absolutely rejects the possibility of a primary tuberculosis of the middle ear, and considers every otitis of tuberculous nature as a secondary manifestation of the general disease. This view is in accord with what is generally accepted in the etiology of tuberculosis elsewhere in the body. A tuberculous kidney or salpingitis, or tuberculosis of the testes, is always a secondary focus to a pulmonary phthisis. Anent the route followed by bacteria from the primary focus to the middle ear, aside from immediate contact with an infected gland, invasion of the tympanum may take place either through the blood or the lymph, or by a direct inoculation of the parts. Cemach has shown that infection of the ear in tuberculosis takes place most frequently during the second anaphylactic stage of the disease and occurs by way of the circulation. The fewest of his cases showed binaural localization. In those that did, he concluded that the bacteria traveled directly through the eustachian tube (inoculation metastasis).

It is possibile that the occurrence of tuberculous otitides is less common in this country than in Europe. Cemach [10] states that every fourth or fifth otitis media in childhood is tuberculous; while in

9. Bussacca, G.: A Bacteriological Research of the Chronic Suppurative Otitis Media, with Special Regard to the Presence of Vincent's Organisms; the Results of Treatment with Neoarsphenamin, Arch. ital. di otol. **34**:414, 1923.

10. Cemach, A. J.: Tuberculosis of the Ear in Modern Tubercular Research, Monatschr. f. Ohrenh. **58**:253 (March) 1924; ibid, **58**:334 (April) 1924.

adults one in twenty is specific in nature. The suspicious elements fall into three groups. There are those which are definite evidence of tuberculous otitis, such as a nodule on the membrana tympani which rapidly disintegrates, causing a total destruction of the drum; exposed bony rough surfaces in the middle ear; grayish discoloration of the exposed mastoid wound and slow healing; and a tendency on the part of the wound to break open repeatedly and cast off sequestrums. In the second division are those factors which, while not diagnostic, are nevertheless suspicious. They consist of the gradual appearance of a circular stenosis of the bony part of the canal, of the painless development of a fistula in the mastoid region or in the ear itself and, in acute cases, of an increased proliferation of granulation tissue. The third group includes such other factors as positive tuberculosis of the periauricular glands, tuberculosis of the lungs, multiple perforations of the membrana tympani, and paralysis of the facial nerve in chronic otitis. These last are to be interpreted only in connection with symptoms of the other groups; in themselves they are not diagnostic. In addition, Cemach advocates the use of the specific focal reaction in the diagnosis of aural tuberculosis. He employs the subcutaneous injections of tuberculin except when there are progressive lesions of tuberculosis anywhere in the body. The focal reaction in the ear is synchronous with the cutaneous and general reaction. A hyperemia of the drum and the tympanic mucosa appears. Other signs of acute inflammation may also be evident, such as pain in the ear and hypersecretion. Cemach considers that a positive focal reaction is absolute proof of the tuberculous etiology of otitis media, but that a negative reaction should not be considered as ruling out tuberculosis of the ear.

These figures of Cemach are indeed alarming. One is almost led to believe that in this country we are overlooking the etiologic importance of tuberculosis in aural disease. Certainly the percentage of tuberculous otitis reported in the American literature is almost negligible. It is possible, of course, that improved sanitation and hygiene here, coupled with the untiring endeavors of various organizations for the prevention of tuberculosis, have either lessened the incidence of the disease or have been instrumental in causing an earlier diagnosis of general tuberculosis, thus preventing the onset of secondary lesions in the ear. The institutions in which tuberculous patients are treated may be able to furnish statistics of aural tuberculosis in the United States. A survey of that kind would indeed be instructive.

The case reported by Keiper [11] and those by Webber [12] are of interest in that they apparently present an acute suppurative otitis media, due

11. Keiper, G. F.: Primary Diphtheria of the Middle Ear, Laryngoscope **34**: 426 (June) 1924.

12. Webber, R.: Klebs-Loeffler Infection of the Middle Ear and Mastoid, Ann. Otol., Rhinol. & Laryngol. **32**:1246 (Dec.) 1923.

to the Klebs-Loeffler bacillus, primary in the middle ear. Why the diphtheria organism should fail to produce its wonted reactions in the throat and cause a distinct reaction in the ear cannot be explained. In any event, it is certainly advisable to culture all middle ear discharge. While in the great majority of instances the result of such examination would have little bearing on the treatment or outcome of the case, in those patients in whom the aural discharge is but a local manifestation of a general disease, specific treatment would result in speedy recovery.

BACTERIA AS A SURGICAL INDICATION

It remained for Boyd-Snee [13] to advocate surgical intervention on the mastoid in suppurative otitides, in which streptococcus was demonstrated to be present in the ear discharge. Valentine and many others have reported streptococci in the aural discharge of patients whose middle ear lesion cleared and in whom recovery took place without resort to surgery on the mastoid process. The otorrhea in the large majority of acute middle ear infections recently studied by this author gave streptococcus organisms; most of them cleared up without any sequelae and without operation. It is only in that type of otitic systemic infection termed the hemorrhagic mastoiditis (this will be taken up later on), in which *Streptococcus hemolyticus* causes a distinct clinical and pathologic syndrome, that agreement exists with the findings of Boyd-Snee. Probably all of the latter's cases were mastoiditis of this type produced by the hemolytic streptococcus. As a general rule, we would be most cautious in taking the finding of a streptococcus in the aural discharge as a definite indication for mastoid surgery.

Urbantschitsch [14] presents a series of ten cases which have the following features in common:

1. An acute onset.
2. Scant or absent otorrhea.
3. A whitish-pink suffusion of the membrana tympani with no evidence of perforation.
4. No characteristic otoscopic picture of acute otitis media of suppurative nature.
5. The finding of free viscous pus in the cellular structure of the mastoid process.

He terms these cases the "mucosusotitis type." Their clinical course resembles in every respect that of cases which have a *Streptococcus*

13. Boyd-Snee, H.: An Otologic Study of the Streptococcus Organism, Ann. Otol., Rhinol. & Laryngol. **33**:929 (Sept.) 1924.

14. Urbantschitsch, E.: Acute Otitis Media of the Type of Mucosus Otitis Without the Demonstration of Streptococcus Mucosus, Monatschr. f. Ohrenh. **58**:594 (July) 1924.

mucosus-capsulatus infection of the middle ear; but there is no evidence of that organism's presence in either culture or smear in Urbantschitsch's report.

While *Streptococcus mucosus-capsulatus* (pneumococcus Type III) produces certain characteristic reactions in the tissues, on the whole the symptom complex resulting from its presence is the same as that caused by any other invading organism. On determining its presence, however, the aurist must bear in mind its potential sequelae and should consider the possible necessity for operation, even though the clinical manifestations may not seem very threatening. When these patients do come to operation, the pathologic changes exhibited in the middle ear and the mastoid process will be found in no way different from that produced by other pathogenic streptococci. The danger of the so-called *Streptococcus mucosus-capsulatus* (pneumococcus Type III) lies in the fact that this organism, possessing a capsule, does not permit the body to develop barriers against its further invasion of the system. Then, because of the lack of a protective reaction on the part of the body, it is able to advance to the more vital structures without giving signs of its progression.

Kettlekamp [15] describes three cases of what he too calls mucosus-otitis, in which *Streptococcus mucosus-capsulatus* was found in the aural discharge. The clinical pictures here are similar to those described by Urbantschitsch, but in these cases the organism is definitely identified. It has long been known that certain conditions, among which diabetes is not infrequently found, predispose to a variety of mastoid disease which has been termed the "painless type" of coalescent mastoiditis. The "painless type" is often found when the lesion is produced by pneumococcus Type III (*Streptococcus mucosus-capsulatus*) or by the Friedlander bacillus. As far as pathology is concerned, this lesion shows nothing by which it can be differentiated from the ordinary painful (classical) type of acute coalescent mastoiditis. The essential difference between the two lies in the presence or absence of pain. The "painless type," which approximates the cases observed by Kettlekamp and Urbantschitsch, is often characterized by an absence of all pain sensations, whether spontaneous or elicited. Another important distinction lies in the fact that patients of the "painless type" frequently present a persistent aural discharge, which is out of all proportion to the apparent well-being of the patient. These cases are more properly grouped, not under the heading of diabetic mastoiditis or mucosusotitis, but under the pathologic designation of acute coalescent mastoiditis of the painless or asymptomatic type. In this way one embraces all cases such as are described by Urbantschitsch and Kettlekamp and which present similar clinical and pathologic pictures.

15. Kettlekamp, F. O.: Mucosus Otitis, Laryngoscope **34**:44 (Jan.) 1924.

In this grouping would also come the cases described by Bertein.[16] He describes these as latent mastoiditis occurring in the course of acute otitis media. The patients presented none of the wonted signs of mastoiditis. There were no local areas of tenderness and no constitutional symptoms until suddenly phenomena of grave import supervened, and the systemic signs of a general infection were manifested. These cases cannot logically be considered as having had a latent lesion. It is hardly scientific to consider a purulent lesion as latent. The disease may be asymptomatic for a period in its evolution. It may be painless and its nature may be such that no constitutional signs are apparent to signify its existence. But the presence of a suppuration in the ear implies an active infection. Therefore, in the interests of scientific accuracy, it is better to class cases like those of Bertein as belonging to the painless, asymptomatic type. When the final stage of their clinical course is reached, the signs of general sepsis are comprehended. On the operating table such patients usually present the lesion designated as acute coalescent mastoiditis.

OTITIS MEDIA IN INFECTIOUS DISEASES

In reference to the occurrence of otitis media in the exanthematous diseases, the statistical and experimental reports of Harries and Gilhespy[17] and of Gardiner[18] are instructive. The former, referring especially to otitis in scarlet fever, believe that the severity of the middle ear infection is distinctly lessened if the adenoid tissue is removed. This can be done with safety during the fourth week of the disease. In twenty patients in whom the adenoid tissue was untouched, the average duration of the otitis was 32.4 days. In fifty patients in whom the adenoids were removed, the average duration of the middle ear infection was 15.8 days. The authors state that mastoid complications were rare in their experience. Gardiner, on studying measles in relation to otitis, comes to the same conclusion, namely, that the removal of the adenoid tissue in these cases hastens the cessation of the aural discharge. He also observes that in patients in whom adenoid tissue had been removed prior to the onset of the measles, the infection was of a much milder character and responded readily to conservative therapy. He concludes that uncomplicated measles does not produce so severe an otitis as scarlet fever, and that in measles the discharge in the tympanum is confined to the mucous membrane, as evidenced by the large number of central perforations.

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16. Bertein, P.: Latent Mastoiditis in the Course of Acute Otitis Media, Presse méd. **32**:841 (Oct. 25) 1924.

17. Harries, E. H. R., and Gilhespy, F. B.: Scarlet Fever Otitis, Lancet **1**:843 (April 26) 1924.

18. Gardiner, W. T.: Otitis Media in Measles, J. Laryngol. & Otol. **39**: 614 (Nov.) 1924.

These observations on the effect of the removal of adenoid tissue on the severity of otitides following the exanthems are important. In all otitis media conditions due to scarlet fever and measles is found a tendency to extensive involvement of the bony structures in the middle ear and mastoid process. Perhaps the secret in the prevention of this type of lesion following those diseases lies in the early application of the methods advocated by Harries, Gilhespy and Gardiner. Certainly the comparison of their results in patients in whom the adenoids were removed with the outcome in unoperated cases seems to offer convincing proof of the value of adenoidectomy performed before the period of quarantine has passed.

Zeidler [19] asserts from the observation of his cases that typhoid fever is always accompanied by an aural complication. Typhoid lesions of the internal ear were found in every case, according to his statement, but only in 7 per cent. was there an associated middle ear suppuration. These findings of definite disease in the inner ear are interesting, because the general conception has been that typhoid fever affects the hearing by injuring the acoustic centers in the temporosphenoidal lobe. The infrequency of middle ear suppuration in connection with typhoid fever is also worthy of note.

(To be Continued)

News and Comment

AMERICAN BOARD OF OTOLARYNGOLOGY

An examination was held by the American Board of Otolaryngology on May 26, 1925, at the Medico-Chirurgical Hospital, Philadelphia, with the following result: passed, 137; failed, 20; total examined, 157.

The next examination will be held at the University of Illinois School of Medicine on Oct. 19, 1925. Applications may be secured from the secretary, Dr. H. W. Loeb, 1402 South Grand Boulevard, St. Louis.

LOCAL SOCIETY MEETINGS

PHILADELPHIA: Section on Otology and Laryngology of the College of Physicians of Philadelphia meets on the third Wednesday of each month from October to June, at 8:30 p. m., at the College of Physicians. Chairman, Dr. Fielding O. Lewis; Secretary, Dr. James A. Babbitt, 2117 Chestnut Street.

CLEVELAND: Ophthalmological and Otolaryngological Section of Cleveland Academy of Medicine meets on the fourth Friday of each month at the Winton Hotel. Chairman, Dr. E. W. Garrett; Secretary, Dr. M. Paul Motto, Rose Building.

CHICAGO: Chicago Laryngological and Otological Society meets on the first Monday of the month from October to May at the Crystal Room, Great Northern Hotel. Chairman, Dr. E. H. Norcross; Secretary, Dr. A. A. Hayden, 25 East Washington Street.

MILWAUKEE: Milwaukee Oto-Ophthalmic Society meets on the third Tuesday of the month, at the University Club, at 6:30 p. m. President, Dr. John E. Guy; Sec.-Treas., Dr. John J. Brooks, Majestic Building.

KANSAS CITY, MO.: Kansas City Eye, Ear and Nose Society meets on the evening of the third Thursday of each month from September to May, at the Baltimore Hotel. The afternoons of the third Thursday of November, January and March are given over to clinical work. President, Dr. James May; Secretary, Dr. A. E. Eubank.

Abstracts from Current Literature

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THE FUNCTION OF THE VESTIBULAR ORGAN AND THE CLINICAL EXAMINATION OF THE OTOLITHIC APPARATUS. F. H. QUIX, J. Larygnol. & Otol. **40**:425 (July) 1925.

The purpose of this paper is to demonstrate the importance and method of examination of the otolithic apparatus. First, a survey of the anatomy, topography and function of the otolithic apparatus is given. The utricular maculae preside over movements of the head in the frontal plane and the saccular maculae over movements in the sagittal plane. Stimulation of these maculae calls forth reflexes of the eyes and body musculature which serve to maintain the normal equilibrium of the head and body.

Then follows a survey of the anatomy, topography and function of the semicircular canal system, and it is pointed out that each pair of canals is stimulated by accelerated movement in its own plane and calls forth prompt movements of the eyes and body musculature which serve to protect the body against any sudden change of position. (To be concluded.)

TUBERCULOUS DISEASE OF THE MIDDLE EAR. H. D. D. M'CART, J. Laryngol. & Otol. **40**:456 (July) 1925.

This paper is based on the analysis of the case records of tuberculous otitis media of the Royal Infirmary, Edinburgh, and the private cases of Dr. J. S. Fraser for a period of ten years (from 1914 to 1924). A careful study is made of the age incidence and a comparison made with the report of Drs. Logan Turner and Fraser for the previous ten years. The etiology, clinical aspect, operative findings and pathology are discussed and a concise résumé is given.

In the present series, the maximum age incidence was from 2 to 5 years. The infection is usually milk-borne and therefore the bovine type of tubercle bacillus is the more common. The route of infection to the ear is either via the eustachian tube or blood stream.

McNALLY, Montreal.

MALFORMATIONS OF THE COCHLEA AND HEARING. F. R. NAGER, Ztschr. f. Hals-, Nasen-u. Ohrenh. **11**:149 (June 2) 1925.

Nagel adds two cases to the literature. He summarizes as follows: Two cases of familial hereditary hardness of hearing are reported, which showed the complex of symptoms of an inner ear hardness of hearing when they were examined during life. Histologic examination revealed a malformation of the labyrinth. In the first case there was an underdevelopment of the cochlea with a reduction of the number of turns. The second case showed the picture of congenital deafmutism of the type of Modini, with dilatation of the cochlear channel and of the sacculus endolymphaticus and atrophy of the sensory cells. Both cases confirmed the presence of considerable rests of hearing even if the malformations of the labyrinth were strongly developed. The functional test showed a reduction of the lower tone limit. Corresponding with this finding, a defect of the upper portion of the cochlea was found in both cases. In order to elucidate the pathogenesis, other analogies of labyrinthian con-

genital and acquired changes are cited and the respective causes mentioned which might come into consideration concerning the development of the described malformation.

THE INFLUENCE ON THE ACUITY OF HEARING BY CALORIC IRRITATION OF THE INNER EAR AND ALSO BY VALSALVA'S AND MUELLER'S EXPERIMENT. E. LUESCHER, Ztschr. f. Hals-, Nasen-u. Ohrenh. **11**:184 (June 2) 1925.

Valsalva's experiment is carried out in a different way from that used by otologists. The subject inspires deeply and then makes a maximum effort of expiration with a closed glottis. Air by this method is not pressed into the tympanic cavity and does not affect the sound conduction. The effect of Valsalva's experiment on circulation has been investigated. It results in a considerable congestion of the veins and capillaries with correspondingly increased blood pressure. In the beginning of the experiment, the arterial pressure is increased by 70 mm. of mercury. Toward the end of the experiment the arterial pressure decreases rapidly. Therefore the effect of Valsalva's experiment is a great congestive hyphemia in the realm of the veins and capillaries.

Mueller's experiment consists of a forced effort to inspire with a closed glottis after a complete expiration. The region of the veins and capillaries is emptied toward the heart and the interior of the thorax, and a sinking of the pressure and hyphemia occur in these regions.

1. *Valsalva's Experiment.*—Four persons were examined concerning the upper tone limits and the acuity of hearing for 10,000, 5,000 and 3,000 vibrations. In all, slight changes occurred in both directions which were all covered, however, by the limits of error which had been established before. At the same time, sometimes slight noises of a deep roaring nature were noticed in the ear. It can be excluded that a considerable influence of the Valsava experiment exists concerning the upper tone limit and for the acuity of hearing for high tones during the duration of the experiment or for a short time afterward.

2. *Mueller's Experiment.*—Of the four cases one showed a slight diminution of the hearing limit and of the acuity of hearing for 10,000, 5,000 and 3,000 vibrations within the limits of error. Sometimes slight deep roaring noises were noticed. An influence on the upper tone limit or on the acuity of hearing for high tones could not be established.

The Caloric Irritation

Name	Before	After	Difference
H. B.	16,800	16,820	— 60
V. W.	16,570-16,510	16,510	— 30
M. K.	16,760-16,570	16,660-16,510	— 80
B. L.	16,880-16,660	16,660-16,620	—130

The table shows that the upper tone limit is diminished in all cases from 30 to 130 vibrations by cold irrigation. This change, however, comes within the limit of error. An influence on the inner ear cannot be established with certainty. Subjective noises were noticed only to a limited degree and can be attributed to the middle ear disturbance.

Conclusions: 1. In people with healthy ears, an essential change in the acuity of hearing for high tones cannot be induced by the Valsalva experiment

(hyphemia of the inner ear and central nervous system), nor by Mueller's experiment (anemia of the inner ear and of the central nervous system).

2. The acuity of hearing for high tones in people with healthy ears remains unchanged during cold caloric irritation.

3. It is evident, therefore, that the mechanical movement of disturbance of circulation, of a short duration, in the inner ear influences the function of the cochlear portion not at all or only insignificantly and that in hearing disturbances in consequence of such disturbances of circulation the chemical factor, namely, the disturbance in nutrition, comes to the fore. The beforementioned experiments cannot determine whether in circulatory disturbances of a long duration the mechanical factor may cause disturbances in hearing by tissue changes. A direct effect of pressure is excluded in these cases.

4. The same considerations apply to the effect of circulatory disturbance on the central nervous system.

THE SPOT OF EXCITATION OF THE LABYRINTHIAN FISTULA SYMPTOM. AN ANSWER TO S. H. MYGIND. G. V. T. BORRIES, Ztschr. f. Hals-, Nasen-u. Ohrenh. **11**:206 (June 2) 1925.

Borries claims that his and Mygind's theory are not identical. Borries is of the opinion that the fistula symptom is a labyrinthian reaction, elicited from the cristae and the maculae, like the caloric reaction, according to his theory. Mygind thinks that the fistula symptom can only emanate from the otolith organs. The main point of the controversy according to Borries is that Mygind denies that Borries could have understood a "general labyrinth reaction," when speaking of a reaction of the cristae as well as of the maculae. Borries claims that it must be understood that a nystagmus is elicited by general vessel changes of the labyrinth.

CHANGES OF THE AIR PASSAGES AND OF THE EAR IN POLYCYTHEMIA. WILHELM SCHREYER, Ztschr. f. Hals-, Nasen-u. Ohrenh. **11**:209 (June 2) 1925.

Polycythemia frequently causes Ménière's complex of symptoms and changes of the mucous membranes of the air passages in a typical manner. The disease is marked by a peculiar redness of the skin, by an increase of leukocytes and erythrocytes. Frequently the numbers are trebled. In the Vaquez type, an enlargement of the spleen, in the Geisboeck type, increased blood pressure is found. Lately endocrine and constitutional disturbances are considered to be the cause of this hematopoiesis. All vessels are enormously widened. This dilatation reaches the smallest arteries, veins and capillaries. The author describes two cases. In the first case typical attacks of dizziness had occurred. Frequently considerable hemorrhages after small interferences are reported, that is, after extraction of a tooth. Death frequently results from apoplexy, hemorrhages into cavities, etc., although the blood coagulation time was mostly normal. The attacks of dizziness, such as occurred in the first patient, occur frequently in polycythemia. They are often combined with headache, tinnitus or hardness of hearing. Vomiting and syncope also occur. This is the typical picture of Ménière's symptom complex. Schreyer mentions a third patient who was observed by Stern and examined otologically by Hinsberg. Since October, 1905, the patient, a middle aged man, had attacks of dizziness; at these occasions he collapsed and the skin was covered with cold perspiration. He was obliged to lie down quickly because everything

in the room moved around. The patient vomited as often as 30 times a day and then he was dazed. After these attacks the patient felt weak. The attacks recurred at intervals of one to three weeks. Since the summer of 1906, he heard a shrill tone and ringing in the left ear. Hardness of hearing set in. The number of erythrocytes was 7,120,000, but no tumor of the spleen was present. In the fall of 1906, the upper tone limit was shortened by almost two octaves, the lower tone limit was intact. Rinné test was positive on both sides. The whispered voice was heard at a distance of 1 m. in the left ear. The diagnosis of a labyrinthian disease with Ménière's complex of symptoms on the left side was made. Stern had observed several other patients with similar symptoms. In a patient, aged 24, who became suddenly dizzy, vomited and suffered from headache, the acuity of hearing for whispered voice was diminished, the upper tone limit had decreased on the left side and nystagmus to the right was present. The goniometric examination pointed to a disease of the left side. In one of the patients Professor Brieger noticed a great congestion of the vessels of the drum membrane and of the hammer handle. It is interesting to state that the attacks of dizziness in the first patient could be bettered and for a time be cured by repeated venesections. No doubt there is a dilatation and congestion of all vessels of the inner ear analogous to the general dilatation of vessels in the body. There is also great hyperemia of the brain in polycythemia and increased pressure of the cerebrospinal fluid which develops according to Boettner in consequence of the disproportionate relation between the enlarged brain and the interior of the skull. The clinical investigations of polycythemic patients may perhaps be able to throw new light on the theory of the vasomotor nystagmus of Kobrak.

Concerning the New Formation of Nerve Fibers in the Vestibulum and Semicircular Canals (Vestibularisneuroma). H. Haike, Ztschr. f. Hals-, Nasen- u. Ohrenh. **11**:213 (June 2) 1925.

In 1870, Schwartze observed a new formation of nerve fibers in the end region of the vestibular nerve. Three decades later, Schwabach observed another case (bilaterally). Both authors considered the formation an analogon to an amputation neuroma. Other cases were reported by Lange, Brock (three cases) Mayer (two cases) and Zange. Haike reports the findings in the ears of a patient, aged 60. When the patient was 20, she had suffered from cholera and became hard of hearing in both ears. Some time later an ear suppuration set in in the right ear, which continued with short intermissions. Toward the end of 1910, she was admitted to the clinic on account of headache and dizziness.

Examination: On the right side there was total destruction of the drum membrane. The Weber test was localized to the left. The Rinné test on the right side was negative, on the left side positive. The whispered voice was not heard on the right side, on the left side it was heard at a distance of 1.4 m. Bone conduction was shortened on the right side and lengthened on the left side. Nystagmus was present on both sides. The mastoid process on the right side was very tender on pressure. On March 7, 1911, a radical operation was performed. There were granulations in the oval window; the stapes were missing; fistula was found on the horizontal semicircular canal. After the operation the patient felt better but the symptoms did not disappear entirely. They were aggravated during the ensuing months. In October, the wound cavity was found well epidermatized. There was spontaneous nystagmus to the left. The

caloric and turning reactions were found to be missing on the right side during repeated examinations during several months. The neurologic finding pointed to an abscess of the cerebellum. April 29, 1912, a preliminary operation for the formation of an osseous flap was done, and the patient died the next day. Necropsy revealed extensive softening of the right cerebellar hemisphere and a recent hemorrhage from an aneurysema of the posterior inferior cerebellar artery which had burst. The microscopic examination of the inner ear revealed, among other things, extensive proliferation of nerve fibers embedded in connective tissue, which represented themselves as partly convoluted and entangled small tumors, as they have been described by Schwabach, Lange and Brock.

Mayer's conjecture has been contradicted by the clinically examined cases of Brock. Mayer had assumed that the function (fistula symptom, caloric reaction) which reappeared after labyrinth suppuration, labyrinth operation could be explained by such a new formation of nerve fibers in the vestibulum or in the semicircular canals.

Urbantschitsch and Bondy have been the first to call attention to such a reappearing function. Haike's patient had been examined repeatedly before and after the labyrinth operation and did not react to the caloric or turning tests. Of the thirteen neuromas of the vestibularis seven had followed meningitis and cerebrospinal meningitis respectively (one case of Schwartze, one case of Schwabach, bilaterally, two cases of Brock, bilaterally), whereas examination of hearing organs of patients suffering from suppurative middle ear labyrinthian processes showed a neuroma comparatively seldom, namely, six times. This can be explained by the fact that meningitis and cerebrospinal meningitis attack the ear (and especially the labyrinth) acutely and destroy the nerve structures in a very short time so that a weakening of the vitality of the tissues does not occur as it does in a chronic suppurative process. Furthermore, in all cases of neuroma of the vestibularis the beginning or the whole course of the disease took place in childhood or in youth when the power of the regeneration was especially great. Brock is of the opinion that the vestibular nerve is more resistant than the cochlear nerve and that therefore the cochlear nerve does not form a neuroma. Haike, however, is of a different opinion. He thinks that the spiral ganglion perishes in such extensive inflammatory processes, whereas the vestibular ganglion which is separated by the lamina cribrosa does not suffer. The trophic irritation emanating from the ganglion persists, but it is missing for the end fibers of the cochlear nerve and this trophic irritation is necessary for the regeneration.

CONCERNING THE ORIGIN OF CHOLESTEATOMA OF THE MIDDLE EAR. W. LANGE, Ztschr. f. Hals-, Nasen-u. Ohrenh. **11**:250 (June 2) 1925.

Lange comes to the conclusion that a cholesteatoma can develop after any inflammation in the tympanic cavity. The epidermis may immigrate through large perforations of the drum membrane and epidermatize the mucous membrane which is denuded of epithelium. It may also immigrate without any perforation of the drum membrane if there is no resistance in the membrana flaccida. Atypical exuberant growths of epidermis are based on a proliferating inflammation. If a cholesteatoma has been formed under these conditions, an entirely intact drum membrane may remain after the releasing inflammation has healed (cases of Manasse). A certain individual disposition of the epidermis must be considered. The energy of the growth of the epidermis varies according to age and unknown general constitutional factors. Cholesteatomas will develop more easily in individuals with a strong epidermis. The observations after radical

operations agree with this. It is generally observed that after operations for cholesteatoma the bone cavities epidermatize well and more quickly, even if all epidermis is removed from the middle ear cavities. After rarefying bone diseases without cholesteatoma, the epidermatization proceeds much more slowly also if the walls of the cavity are formed by firm bone after an operation. The cholesteatoma is the expression of a healthy constitution of the epidermis. It is not surprising that cholesteatomas may develop in various ways. In some instances Lange found a formation of cholesteatoma in the middle portion of the tympanic cavity into which the epidermis had grown through a large perforation, beside a cholesteatoma which had developed from the membrana flaccida. It was entirely separate. Pneumatization furthers the growth of cholesteatoma. Its effect is confined to the age of pneumatization, the first years of life. Lange agrees with Wittmaack. If the cholesteatoma develops during the first year of life it encounters in the middle ear cavities a physiologic process which represents a breaking up of the bone by the subepithelial tissue, the first act of pneumatization. The growth of epithelium is very vivid at this time and the second act of pneumatization, the secondary wall formation of the cell is suppressed through the rapid growth of the epidermis cyst. Rapid development of large cholesteatomas must result during the first years of life. The surprising extent of cholesteatoma in infancy which has repeatedly led to consider them to be congenital tumors, can be explained by assuming an inflammatory genesis which is supported by normal pneumatization. A chronic suppuration is not the condition sine qua non for the formation of a cholesteatoma. The drum membrane may be intact if a proliferating inflammation of a longer duration is present. The cholesteatoma is a cystic or cystlike growth of epidermis on an inflammatory basis. All this does not exclude that real tumors, for instance, carcinomas, can develop from these pathologic formations of epidermis. General experience points to the fact the squamous celled epithelioma can develop on the basis of a chronic middle ear suppuration. The objection that the suppuration is secondary to the carcinoma can, as a rule, be met only with difficulty or not at all. The origin of the development of a squamous celled epithelioma in the middle ear has not yet been reported.

AMBERG, Detroit.

Pharynx

A FEW REMARKS CONCERNING TONSILLECTOMY. STRUYCKEN, Ztschr. f. Hals-, Nasen-u. Ohrenh. **11**:181 (June 2) 1925.

Struycken combines several well known instruments and describes a new combination of a suction mouth spatule ("Sauge-Mundloeffel"). In tonsillectomy the tongue is pressed downward and the blood is sucked simultaneously. After the upper pole is somewhat loosened from its niche it is not necessary any longer to depress the tongue, but the tip of the spatule is carried into the niche. The upper pole is pressed downward or it is fixed by suction at the upper opening of the suction spatule. The right hand remains free for the use of the cutting or loosening instruments. The stringlike connections with the capsule can be seen more plainly than by using the other methods, and they can be severed at the tonsillar side. The instrument can also be used to advantage not only in surgical procedures but also in the simple spatula laryngoscopy according to Bruening, Benjamin and others. In this case a modification of the cap advised by Kirstein can be used. In a similar manner one can proceed also in hypopharyngoscopy and upper esophagoscopy. If a

blowing apparatus is used instead of a suction pump, ether-chlorethyl or chloroform narcosis can be used intrabuccally or tracheally. If a hemorrhage occurs after tonsillectomy or in the other places which can be reached only with difficulty, the bleeding spot can be detected after removal of the clot and by suction of the blood. The bleeding spot is touched with a little alypin-epinephrin and a small ball of the vaseline gauze is applied for a few minutes under light pressure. Hydrophil gauze strips 5 by 10 cm. are saturated in the autoclave for one hour. The cup is taken from the autoclave and the superfluous vaseline is removed as soon as the vaseline has just congealed. Struycken was only rarely obliged to catch a spurting vessel with an artery forceps and compress it for a little while. It may be surmised that such a vaseline gauze sponge works so well because the thrombus does not adhere to the sponge and is not carried away when the sponge is removed. The fine emulsion of vaseline and blood favors the coagulation of the latter even in hemophils.

THE CENTERS OF PRODUCTION (LEISTUNGS MITTELPUNKTE) IN THE TONSILS OF ADULTS IN TWENTY CASES OF RECURRING ANGINA IN WHICH THE TIME OF THE LAST ATTACK WAS KNOWN, AND AN ANALYSIS OF THE REST OF THE TISSUE CONDITIONS. K. A. HEIBERG, Ztschr. f. Hals-, Nasen- u. Ohrenh. **11**:244 (June 2) 1925.

There is no reason to attribute a deciding factor to the condition of the surface alone concerning the delay of the healing processes. The lymphoid tissue must also be analyzed. The question arises, of what importance the cessation of the function of the secondary nodule is. The method has been published extensively in the *Acta otolaryngologica* **7**, 1924, and in *Virchows Arch. f. path. Anat.* 253, 572, 1924. Increase in lymphocytes was called simple hyperplasia. It is better to look at this increase of lymphocytes as inflammatory reaction. Filtration takes place in the lymph glands, also a weakening of the virulence and immunization. A similar process can be assumed in the lymph tissue of the mucous membrane. Those parts of the mucous membrane which contain adenoid tissue as the tonsils are favored because they themselves contribute to an immunization against an infection. The luminous portion of the secondary nodule (the center of efficiency of the so-called center of germination) has, according to Heiberg, the function to serve as center of destruction for certain lymphocytes. A production of antibodies on the part of the secondary nodules can be understood. If one would follow up, in animals, the development of centers of germination, for instance, by following a smallpox vaccination, one certainly would see a whole series of pictures illustrating this point, pictures which in principle would correspond with the appearance of a tonsil after tonsillitis. First, a relative overloading with small lymphocytes, then a hyperplasia and new-formation of "centers of germination" (centers of production) with strongly developed phagocytosis, under the influence of which the numbers of lymphocytes gradually disappear. Afterward, a part of the "centers of germination" disappears in order to allow again the ordinary development of tissues. In recurring attacks of tonsillitis, the centers of germination suffer and regeneration becomes faulty. The before-mentioned considerations illustrate the question of the usefulness of an enucleation of the tonsil aside from the possible mechanical importance. As a rule not a healthy defense organ is removed, but frequently the removed organ is the seat of a necrobiosis which is of consequence and is the seat of a faulty reaction.

AMBERG, Detroit.

Larynx

CONCERNING TREATMENT OF STENOSES OF THE LARYNX BY STRETCHING. E. BUECH, Ztschr. f. Hals-, Nasen-u. Ohrenh. **11**:177 (June 2) 1925.

The following methods must be considered. 1. Purely surgical treatment. 2. Stretching with the obturator cannula ("Bolzenkanuele"). 3. The combination of surgical and mechanical aftertreatment. 4. Stretching with a soft rubber tube. The choice of treatment depends on the nature of the individual case. Buech concludes that the enormously great variety of stenoses of the larynx easily shows that one single instrument like Brueggemann's cannula cannot be found efficient in all cases. This was experienced in one of Buech's patients when the obturator ("Bolzen") slipped. In this case the stretching was not so far completed that the obturator could have been omitted. As an auxiliary measure Buech was obliged to use the "necktie." (Muck's necktie consists of a metal structure covered with leather. It rests on the shoulders and has an opening in the front, in which the cannula rests. By the aid of a screw-arrangement, the desired position can be obtained.) A satisfactory cure was obtained although under difficult circumstances. In both cases a permanent cicatricial stenosis had been present. An active stretching was necessary and not only a keeping open of the parts. While the cartilagenous structure did not offer any complication, the infantile habitus of the larynx in both cases was extremely unfavorable.

AMBERG, Detroit.

Nose

ANATOMICMECHANIC FORMATION OF THE CONSTRUCTION OF THE FACE SKELETON. J. GALPERIN, Ztschr. f. Hals-, Nasen-u. Ohrenh. **11**:226 (June 2) 1925.

Some authors (Vesal, Schneider, Bartholinus, Highmore, J. Mueller, Henle) contend that the nasal accessory sinuses exist for the purpose of making the weight of the skull lighter, just as in birds the tubular bones of the extremities are filled with air. The calculations of Braun and Classen, however, have shown that a mass of spongy bone in the nasal accessory cavities would increase the weight of the skull only 1 per cent. Other authors (Blumenbach, Hilton, H. Meyer) state that the accessory cavities serve the purpose to warm the inspired air. Yet at each inspiration, very little air enters the main nasal cavity from the accessory cavities. Besides, the mucous membrane and the endosteum of the accessory cavities is so thin and poor in vessels that a warming effect is excluded. The third theory (Hilton, Braun and Classen and Paulsen) is, that in the ancestors of man, in animals with a well preserved sense of smell, the accessory cavities contained the conchae, that in man the organ of smell became atrophic and that the cavities remained empty. Bidder, Hyrtl, and Zuckerkandl, however, have shown that in macrosmates, animals with a highly developed sense of smell (Zuckerkandl), no endings of the olfactory nerve were present, and that the cavities are well developed in animals which do not have a sense of smell, whereas in the lower monkeys, in the carnivoras and in children the accessory cavities are little developed or not at all. The fourth theory (Jessen, J. Sylvius, Bartholinus, Voltolini) claim that the accessory cavities give an agreeable timbre to the voice. Yet children have agreeable tuneful voices and the voice of adults without accessory cavities is not different from those with normally developed cavities. The fifth theory (Komendantoff) claims that a spongy bone would conduct inflammatory processes to adjacent parts (the cranium, orbit), whereas an empty cavity

with compact walls would not act in such a manner. Galperin claims that all these theories are faulty in two respects: 1. They assume that the accessory cavities serve a useful purpose. 2. None of these theories treat the question how these cavities developed and how the mechanism of their development and growth works.

Why should mucous membrane enter the bone unless favorable conditions have been furnished for the entrance of mucous membrane into the same? Galperin tried to answer the question of the origin of accessory cavities by considering the most elementary laws of the mechanism and physiology of the growth of the bones.

The mechanical foundation in the development of the skeleton of the face in man and higher animals is furnished by the work of the chewing apparatus. The teeth of the mandibula are pressed with great power on the teeth of the maxilla, by the temporal, masseter and partly by the pterygoid muscles. This power, according to Harry Black and Eckerman, corresponds in children and women to 20 kg., in men to 25, 50, 80, and even 100 kg.; in the teeth of the athlete Leona Dara it reached even 500 kg.

Animals and the prehistoric man used their mandibles as organs of protection and attack; they also were called on to chew hard roots and fruits. Pressure furnished a formative plastic irritation for the bone. When such formative trophic irritation is missing, the osseous structure is resorbed. When the maxillary bone is subject to pressure it becomes broader, in a child especially fast, when it uses more power in chewing. When the bone has reached a certain thickness the law of distribution of power lines becomes evident. According to these laws the os femorale and other tubular bones become hollow, because the power lines are distributed at the periphery, whereas the central parts receive little or no pressure. The physiologist H. Mayer and the professor of mechanics Kullmann have proved this for tubular bones. Speaking of the palate, Galperin claims that the power of pressure becomes less the farther away it is from the hard palate upward, therefore the hard palate grows as Franke has shown, by additions of layer after layer of the bone at the oral surface of the hard palate, whereas at the nasal surface the bone is gradually resorbed, deepened and changed into a groove, because these parts are not subjected to pressure. The greatest pressure of the chewing apparatus is conveyed through the os zygomaticum to the os frontale in the supraorbital region. Here the os frontale experienced a pressure from both sides. This pressure is the greater the more the chewing apparatus is developed in the animal. This fact becomes clear if viewed from the standpoint of mechanics. The more the mental development of man progressed, the more weapons of defense and attack he produced, the more the art of cooking relieved his jaws from the exertion formerly necessary, the less became the pressure on the zygomatic processes. As soon as the bone in the supraorbital region has reached a certain thickness then the law of distribution of power lines pressure, which is the same for all bones, comes into effect, and in the center the bone is relieved of pressure, which fact leads to the formation of a cavity. This applies also to the frontal sinuses. These cavities are separated by a wall. This wall is not resorbed on account of the pressure exercised by the incisors by way of the os vomeris and the lamina perpendicularis of the ossis ethmoidalis. The pressure, however, is very weak and the wall is very thin, so that even a defect may be found. In an ingenious way Galperin follows his theory concerning the other accessory cavities and the skull, and concludes that he has tried to elucidate the fundamental principals of the structures of the skull as caused by some mechanical factors. He finds that the arrangement of the bones of the skull can be explained thereby. If all mechanical forces are taken into consideration which

contribute to the formation of the skull, and if the modus operandi is carefully studied, it is the opinion of Galperin that it would be possible to explain all details in all parts of the skull.

AMBERG, Detroit.

Miscellaneous

MINERS' NYSTAGMUS. A. FREELAND FERGUS, Lancet **1**:1068 (May) 1925.

In the last few years this disease has become more prevalent. In spite of the painstaking research done, the etiology of the disease is still obscure. The author believes that the disease is not confined to the eyes but that there is a more general involvement of cerebral centers. This is supported by the fact that a large percentage of the more advanced cases show diminished visual acuteness, and as there is no call on visual acuity in mining, but rather on field vision, the diminution of acuity is probably a central defect.

The pulse rate is often very high, even as much as 120. Another striking feature is that the attacks of giddiness, pain and nystagmus, are paroxysmal and may recur with any slight exertion. The treatment consists in detecting the milder cases and giving the patients suitable light work. In more severe cases, the patients should be given almost complete rest, preferably in the country under supervision to prevent loneliness. The author believes that more should be done to investigate the pathology of the condition. Necropsy should be performed on any patient who during life was known to suffer from this disease. A striking fact is the almost total absence of the disease in America, despite the fact that many of the mines are very gaseous, and the illumination defective.

MCNALLY, Montreal.

CONCERNING ABDUCENS PARALYSIS IN EXTRADURAL ABSCESS AT THE TIP OF THE PYRAMID AFTER ACUTE MIDDLE EAR SUPPURATION. G. ENGELHARDT, Ztschr. f. Hals-, Nasen-u. Ohrenh. **11**:194 (June 2) 1925.

Engelhardt mentions that Ulrich furnishes the histologic proof that it is possible that in extradural abscesses at the tip of the pyramid, not meningitis but an anatomic lesion in the extradural course of the nerve may be the cause of an abducens paralysis. According to Engelhardt this observation is important, because the part which the abducens plays, even if an extradural lesion of the same is histologically proved, does not become clearer, if at the time of the first clinical examination symptoms of meningitis have been present.

(Uffenorde: *Arch. f. Ohren, Nasen u. Kehlhopf heilk.* **105,** p. 105, Case 1). Here also, the comparison between the anatomic finding and the clinical picture will decide whether the loss of function of the nerve is caused by an extradural or an intradural lesion. Engelhardt is of the opinion that for determining the genesis of abducens paralysis, whether caused extradurally or intradurally, the macroscopic anatomic finding compared with the clinical picture is sufficient. In a former publication (Contributions to Pathologic Anatomy and General Pathology, 71). Engelhardt had described the various paths of the middle ear suppuration to the tip of the pyramid as shown by pathologic-histologic findings. Two cases had been cited which had been observed clinically and neurologically, but only with macroscopic necropsy findings, one with participation of the labyrinth, the other without. It was very interesting to note that in spite of grave and apparently long lasting changes at the tip of the pyramid, an abducens paresis did not occur imme-

diately, but that it was only then noticed when the terminal suppurative meningitis set in fulminatingly. In the second case it did not even occur when symptoms of pyramidal tract infection pointed to a meningeal irritation.

It is very important to know how frequently irreparable changes at the tip of the pyramid effect the nerve. It would seem that the symptoms of abducens paralysis are missing in the great majority of cases of extradural abscess at the tip of the pyramid so long as the soft brain membranes are not affected in any form, and it is present in middle ear suppurations, which heal without any interference. Therefore, the value of the symptoms of abducens paralysis, even if they are considered only in connection with the whole clinical picture, will be uncertain, but the very early appearing trigeminus symptoms become of greater importance if they are correctly interpreted. These symptoms are probably never absent in lethal cases but, unfortunately, they are also present frequently in light cases.

CHLOROMA OF THE MEATUS. STEFAN KREPUSKA, Ztschr. f. Hals-, Nasen-u. Ohrenh. **11**:196 (June 2) 1925.

Chloroma is a tumorlike formation of greenish color. The first case was recorded by Burns in 1821. He found it when enucleating an eye. The name "chloroma" was first used by King ("A Case of Chloroma," 1853), but it had been in use before. The names green cancer (cancer vert) chlorosarcoma, chlorolymphosarcoma and chloromyelosarcomatosis have also been in use. The color is lost in from two to twenty-four hours when the tumor is exposed to the air; it turns a grayish white. Lehndorf found only 123 cases in the literature. Heredity plays no part in its etiology. In half of the cases chloroma occurs at the cranium and, if situated accordingly, the growth produces the chloromatous face, a fearful froghead-like expression. Later, anemia progresses, hemorrhages appear in the skin and mucous membranes and the patient succumbs in from two to six weeks. The diagnosis appears probable if greenish pus flows from the wound, or if other secretions of a green color are present, if the chloromatous face is present and, in an advanced stage, if the blood picture is changed (grave anemia, atypical cell formation, etc.). The prognosis appears to be bad. Therapeutically, nothing can be accomplished. Arsenic, neo-arsphenamin, calomel, roentgenization and radium are useless.

Two views exist concerning the nature of chloroma. It is considered by some to be a tumor formation, others consider it a form of leukemia. Krepuska reports a case in point. A boy, aged 14 years, was admitted to the St. Rochus hospital in Budapest, April 8, 1915. Three months previously, after a headache of two days' duration, the left ear began to ache. This pain lasted for five days. The hearing remained diminished. Three weeks ago the same symptoms occurred in the right ear. Tinnitus was present in both ears.

Examination disclosed that the meatus was narrowed on both sides, probably on account of sagging of the upper wall. There was no secretion in the canal. A facial paralysis existed on the right side especially of the lower branches. A slight abducens paresis was present on both sides. Whispered voice could not be heard on either side.

On April 21, both mastoids were opened. On both sides typical osteomyelitis of the mastoid process was found. There was free bleeding from the bone and the blood appeared extremely hydremic. On April 25, the patient died. The necropsy (Feher) showed greenish, somewhat transparent compact foci on the squamous portion of the right temporal and occipital

bones, on the visceral side of the spinal column, the cavities, in the abdominal cavity, the lungs, the spleen and the dura. On the right side there was a pyothorax.

There are only two cases of chloroma of the hearing organ mentioned in the literature, one by Alexander (lymphomatous diseases of the ear, *Ztschr. f. Heilk.* 1909, No. 27), and one by Koerner (a case of chloroma of both temporal bones, of both sigmoid sinuses and of both orbits. *Ztschr. f. Ohrenh.*, 1896, No. 29.) Koerner's case showed the picture of a phlebitis of the cavernous sinus. Krepuska claims that in his patient the process originated in the bone marrow. The metastases occurred partly along the lymph spaces, partly along the veins. In this manner the fatal metastasis in the pleura and lung occurred.

REMARKS TO THE ESSAY OF PAUNZ. NECROSIS OF THE UPPER JAW IN THE NEW BORN AND NURSLING. O. KOERNER, Ztschr. f. Hals-, Nasen-u. Ohrenh. **11**:272 (June 2) 1925.

In the new-born and in young children the buds of the temporary and permanent teeth fill the spongy bone of the upper jaw, so that there is little room left for a rudimentary maxillary antrum, which is located far back, upward and inward. The spongy bone is sometimes attacked by osteomyelitis. The symptoms (swelling of the face and palate, with or without evacuation of pus, edema of the lid, displacement of the eye outward and upward, discharge of pus from the nose) may easily be taken for consequences of an empyema of the maxillary antrum. Brown Kelly assumes an infection of a dental sac by injury during birth or by wiping the mouth. Koerner operated on six patients with such a condition. The maxillary antrum is not diseased or is only secondarily so. Paunz thinks that at least in his patients the disease emanates from the maxillary antrum. Koerner disagrees with Paunz.

THE IMPORTANCE OF THE SINKING RAPIDITY OF RED BLOOD CORPUSCLES IN OTO-RHINO-LARYNGOLOGY. B. LIBINSOHN, Ztschr. f. Hals-, Nasen- u. Ohrenh. **11**:221 (June 2) 1925.

All authors agree that the sinking of red blood corpuscles depends on the destruction of protein in the organism and that it is parallel with the same. On this fact rests the diagnostic and prognostic significance of this reaction in the gynecologic, tuberculosis and surgical clinics, Libinsohn used the simplified Westergren method. He examined 145 patients and made 200 tests. Acute phlegmonous processes in the ear and in the upper air passages show a strong sinking reaction corresponding to the intensity of the process. The same applies to malignant tumors but not to benign tumors, irrespective of their location or size, namely, to papillomas of the vocal cords and to large fibromas of the nasopharynx. Otosclerosis and ozena do not show any reaction. A tuberculous process of the larynx does not furnish an acceleration of the reaction. It depends on the condition of the lungs. A grave condition of the larynx, however, that is, extensive ulcerations and perichondritis, which required a quick tracheotomy, showed a sinking reaction of from 4 to 10 mm. even if the lungs were normal, but light and medium light processes showed from 23 to 32 mm. when tuberculosis of the lungs was advanced. The same may be applied to tuberculosis of the skin and nasal mucous membrane. Suppurative processes of the nasal accessory cavities show a comparatively small acceleration or none. Libinsohn's sixteen cases showed from 3 to 18 mm. It reached 30 mm. in a few cases on the fourth to eighth day after the operation when the whole

mucosa was curetted, and healing occurred by granulation. Otitis externa furniculosa did not yield a characteristic finding. Variations of 7 to 32 mm. depended on the presence of a simple suppuration of a lymph gland or an extensive suppuration of the subcutis. In violent otitis media before the incision of the drum membrane, the reaction was strong, also in acute mastoiditis especially with subperiosteal abscesses. In the beginning of acute otitis media the sinking rapidity is influenced by the general cause of the disease. Libinsohn quotes remarkable findings in chronic suppurative processes of the middle ear with bone destruction which are accompanied by caries of the tympanic cavity or of the attic, polyp formation, cholesteatoma, etc. He distinguishes two groups.

One group consists of twenty-three cases with an average rapidity of sinking of 8 mm. (only one case with 21 mm., the rest to 15 mm.). There were no processes with grave destruction. Caries was relatively superficial, granulations did not penetrate deeply into the cells of the mastoid process, and did not come in contact with the large blood vessels and the dura. Extensive cholesteatoma formation was not found. A totally different picture is found in more serious complications. The most pronounced sinking was found in patients in whom the suppurative process reached to the sinus wall, independent of the macroscopic changes of the wall and of the thrombosis of the sinus. The rapidity in acute and chronic cases was the same. There were fourteen cases between 23 and 41 mm., average 31.5 mm.

The other group comprises intracranial complication, extradural abscesses, abscesses of the temporal lobe and of the cerebellum, tuberculous and purulent meningitis. There were nine cases with a sinking rapidity of from 13 to 36 mm. (average 26.5 mm.). In reality there was a more rapid sinking in the patient of 13 mm., as he died within two or three days.

Large cholesteatomas which did not reach the sinus and the dura (nine cases) showed from 20 to 32 mm. (average 26 mm.). Perilabyrinthitis and serous labyrinthitis, even if a fistula of the semicircular canal was present, showed a slower sinking of 8 to 16 mm. (average 12 mm.). By virtue of the sinking reaction, Libinsohn has diagnosed repeatedly more serious complications even without other objective symptoms and with normal temperature, and has corroborated his diagnosis on the operating table. The sinking rapidity can facilitate a differential diagnosis between a labyrinthian and cerebellar disease. Libinsohn comes to the following conclusions.

1. The sinking reaction, *per se*, is of no diagnostic value. It only points to a strong destruction of protein in the body and its presence in the blood.

2. This reaction added to the other objective symptoms furnishes important hints concerning the gravity of the process.

3. The sinking reaction in chronic otitis media with marginal perforation is a valuable sign of serious bone destruction which is frequently not noticeable. It calls for immediate surgical interference.

4. It is desirable that further work may be done concerning this reaction in otorhinolaryngology, because the material is not sufficiently large to allow a definite conclusion.

AMBERG, Detroit.

Society Transactions

AMERICAN OTOLOGICAL SOCIETY

Fifty-Eighth Annual Meeting held in Washington, D. C., May 4 and 5, 1925

EUGENE A. CROCKETT, M.D., *President, in the Chair*

SCIENTIFIC SESSION

MONDAY, MAY 4, 9 A. M.

An invitation was given to the guests of the Society to participate in the discussion.

THE PRESENT STATUS OF THE OPERATION OF REMOVAL OF THE STAPES FOR VERTIGO. Presented by DR. EUGENE A. CROCKETT, President.

Most operations for relief of vertigo are severe and present reasons for surgical hesitation. The removal of the stapes from its niche in the oval window is not dangerous, although it requires nice technic and a careful selection of cases. Prolonged and distressing vertigo is often due to inelasticity of the apparatus of the internal ear, caused by adhesions in the middle ear. These conditions often give the most satisfactory operative results. The following cases may be cited.

Case 1.—Mrs. S., aged 66, had excessive vertigo, deafness and tinnitus in the left ear; the left membrana tympani was thickened and retracted. At operation, the left stapes was removed, with complete relief from vertigo.

Case 2.—Mrs. H., aged 58, had been deaf in the right ear for twelve years, which was worse for eighteen months prior to examination. For five months the patient had had vertigo, nausea and vomiting; she staggered to the right when walking. The caloric reaction was slow and shortened in the right ear and the hearing was decreased. At operation, the stapes of the right ear was removed; no vertigo occurred.

Care should be taken to exclude cases of otosclerosis, patients with tumor, of the eighth nerve also those with cerebral syphilis, locomotor ataxia or multiple sclerosis.

The after results of this operation are excellent in well chosen cases, which are often the type most intractable to ordinary methods of treatment. These symptoms are so incapacitating to the patient that some simple method of relief is most desirable. In the hands of a trained aurist there should be 75 per cent. of recoveries. The only complication to be noted is the danger of causing facial paralysis by accidental lesion of the seventh nerve.

THREE CASES OF OTITIC BRAIN ABSCESS. Presented by DR. S. MACCUEN SMITH, Philadelphia.

The early stage of this disease is usually ushered in by well-known symptoms such as pain, nausea, projectile vomiting with decrease or cessation of otorrhea, but some cases are atypical and misleading as to diagnosis. Some valuable diagnositic signs are: a change in disposition, from brightness to moroseness, or *vice versa;* a lowered, even subnormal pulse rate, temperature

and respiration; yet these do not always occur. Some cases simulate sinus thrombosis and some meningitis.

Case 1.—The first patient in this group was a man, an engineer, first seen in January, 1923, with a history of intermittent discharge of the ear for nine years. If the otorrhea stopped he had intense vertigo and mental confusion. Removal of a polyp from the external canal decreased the discharge and the vertigo. The discharge, however, suggested a necrotic mastoid, and he was advised to have an operation. He agreed, but the operation was postponed for more than a year. Following the operation the patient grew worse and I was called in consultation. I suspected brain abscess. He improved sufficiently to be up and around but later he suffered from diplopia and temporofrontal headache as well as from Bell's palsy. A second mastoid operation relieved these symptoms for two months when he again became ill with symptoms indicating intracranial pressure, but without localizing symptoms. For some time the patient alternated between periods of stupor and comparative mental clearness, but his condition became poorer, so that operation was further postponed. Drainage of the ventricle gave relief from pressure, but the patient died shortly after. Necropsy showed cerebellopontile abscess of the left side.

Case 2.—A man, aged 43, was admitted to the hospital, Sept. 10, 1924. At the age of 7 years, the patient had had acute suppurative otitis media on the right which ruptured, drained for a year, then cleared up. Since then he often removed "wax" from the ear. Thirty years after the first attack, he had influenza, with increase of aural discharge. Four years later he had an attack of severe right-sided head pains, with no discharge or dizziness. The eyes and reflexes were normal. Twenty-four hours later he had chills, fever, a rapid pulse, severe pain, then a profuse discharge occurred which relieved the pain. He returned to work; ten days later he was injured on the head, which caused a return of symptoms. These continued at intervals for a month with increasing severity, until signs of meningitis occurred when he was taken to the hospital, and a diagnosis of brain abscess was made An abscess found in the temporosphenoidal lobe was evacuated through the mastoid wound. He improved, but died of pneumonia two days later. This man probably had had brain abscess for a considerable time. He might have been saved by earlier operation.

Case 3.—A girl, aged 7 years, had had acute otitis media at the age of 4½ years. At 5½ she had influenza with multiple abscesses. Nine months later she developed severe pain in the head for several months, not referable to the ear, until eight months later, when the right membrana tympani ruptured with relief of pain. She then suffered from facial palsy and bulging of the right eye, with projectile vomiting. When admitted to hospital she was semiconscious. A temporosphenoidal lobe abscess was evacuated through the mastoid wound. The child improved markedly for several weeks, when suddenly the symptoms returned. A violent convulsion occurred which terminated in death possibly due to embolism.

TWELVE CASES OF CEREBRAL AND CEREBELLAR ABSCESS. Presented by DR. H. P. CAHILL, Boston.

In all, twelve patients with cerebral or cerebellar abscess were operated on, six by myself and six by other members of the staff. About 60 per cent. of these patients showed infection through the mastoid route. No attempt

was made to operate until the location of the pus was reasonably certain. In two of the cases, late operations were performed; the patients were dying. The abscess had existed for some time and had become well walled off. We ascribe the favorable results in all cases to the use of the Mosher drain, which is made in three sizes, the medium size being the most satisfactory. We use a trocar to locate the abscess and the drain is immediately inserted. It is valuable in acute cases of abscess without a wall as it gives an opportunity for the wall to develop. On account of the cone shaped wire basket which forms the drain, the tendency is for the cerebral tissues gradually to expel it. The drain can be left for weeks and is usually forced out naturally, but we have no technic for its easy removal to avoid fixation by granulations.

DISCUSSION

Dr. Wesley C. Bowers, New York: The fatal cases teach as much as the successful ones. We can diagnose brain abscess sooner if we take more careful histories of patients with chronic discharging ears and also of those with acute purulent otitis media. Some of the postoperative mastoids do not heal well. There is unexplained vomiting with headache. Some patients develop abscesses which become quiescent while we are watching them. More attention should be paid to distinct changes in behavior — tearfulness, loss of self control, listlessness, excitement, depression, visual disturbance and gait disturbance — numerous little signs about which we do not question the patient. I remember one patient who was referred to me for dressings. I asked how he felt, he said he did not feel sick, he had no temperature and no trouble walking, except that he could not raise his right foot. That was the only symptom which gave a hint of brain abscess. Later, his temperature rose and a sphenoidal abscess developed.

If we are careful we can make a diagnosis, but we have to guess at the location very often. When a patient is seen in coma or convulsions, we have to guess where the trouble is. In reading about brain abscess, the most striking fact is its lengthy duration. It seems to me that if patients are carefully tested and examined at frequent periods, the diagnosis would be made more often. It is pathetic to read the story of a patient with brain abscess, who has been watched for a long time with no record made of labyrinthine or vestibular reaction. If the temporosphenoidal lobe has been explored and nothing found the posterior fossa should be explored. Dr. Cahill's results are noteworthy, and he is to be congratulated.

Dr. Edward B. Dench, New York: I would like to congratulate Dr. Cahill on the series of cases which, so far as I know, are unique in the otologic literature, not excluding the remarkable series of Sir William McKenzie, whose results we are inclined to doubt. This work is modern; it is done under our own supervision and reported promptly. With all due respect to the Mosher drain, there must be something more to account for these results. I think early diagnoses and better asepsis have been a contributing factor. I have never used this drain but I think it must be an excellent procedure. This reminds me of a technic of excising the whole abscess cavity, and also of a method of Lemaitre. Lemaitre leaves the angle open and puts in a larger tube each day; the results are similar to those following the use of the Mosher drain.

Dr. Philip Hammond, Boston: I would like to add a word as to the use of the drain. I saw the first case of brain abscess in our community drained by this method, in which the patient survived. The first drain was

of gauze and the patient became markedly worse. He had absolute aphasia and had to have a second operation. He reacted to the second well and a Mosher drain was put in, used in the manner Dr. Cahill recommends. I had a conference with my colleagues because we did not know what to do with the drain. At the end of three weeks the granulation tissue had grown over the drain and we had to dissect it out.

Seeking a remedy for this condition, I think if the drain is molded slightly from a mold now commonly used, namely, that of the Gruber speculum, it could be rotated slightly every day and overcome the resistance of the deeper tissues to the drain. This has worked out very successfully. I think this had a bearing on the last fatal case of Dr. Cahill. If the drain had not been so adherent to the deeper tissues, with evulsion of the pathologic membrane he might have had more complete success.

Dr. C. L. Tobey, Boston: One crucial point is the proper localization of the brain abscess before treating it. These cases emphasize the fact that Dr. Cahill has an unusual knowledge of the pathologic anatomy of the brain.

Dr. A. B. Randall, Philadelphia: It is noticeable in reports of these brain abscesses that so little has been said of Dr. Sloan's work on the early and important symptoms. In view of the necessity of early diagnosis, I think that more attention should be paid to these symptoms.

Dr. Lewis Fisher, Philadelphia: We can receive but little from the neurologists in the diagnosis of doubtful cases. When the nerve findings are plain enough for the neurologists to work on there is little to be done for the patient. To treat these cases successfully, we must make an early diagnosis. In the examination of a large number of brain lesions of various kinds I was struck with the frequency with which the labyrinthine reactions are present in intracranial trouble. One sign is uniformly present, that is, the presence of response to the vertical semicircular canal. I carried out observations on every mastoid case, and the work has more than repaid me for my trouble. Every patient with mastoid trouble not dying will show absence of the vertical canal response on the opposite side. Such a condition is not a simple mastoid. There will be many storms in its course. In many of the cases of suppurating ears reported today there are histories of intermittent attacks of unconsciousness, visual disturbance and temporary paralysis, in which brain abscess should be suspected. The mere performing of labyrinthine tests on the other side would have thrown light on the presence or absence of a brain lesion. This is past the theoretical stage. I do not want to be understood as saying that when this reaction is absent there is brain abscess, but when this phenomenon appears, the case is complicated.

Dr. H. I. Lillie, Rochester, Minn.: In all of these cases there have been neurologic findings, such as choked disk. In using these new methods of drainage, the most important discovery is that there is a favorable time to explore the brain. There is also an unfavorable time, and that is before localization has developed. In our series we have had eight deaths out of seventeen. In the last six cases we have used this method of drainage and found it quite satisfactory.

Dr. Philip D. Kerrison, New York: The results in this series of cases depended on the proper selection of patients for operation, and on Dr. Cahill's skill in interpreting cerebral conditions. However, we are getting away from the effect of technic. I want to suggest the analogy of the cases reported today to the work of Dr. King of Bellevue. He exposes the dura over the

site of the abscess, turning back the flap of the dura. He puts the drain or needle in to demonstrate the presence of the abscess. Then he cuts out a cone of tissue, the basis of the operation being that the intradural pressure will evert the abscess cavity and the cure will come about by a diverticulum. I saw this operation performed. The result looked favorable although that patient died. Four patients, however, made good recovery. I think this technic is a distinct advance on that of eversion of the abscess cavity, as the result is logical. It prevents merely superficial drainage and the obstruction of drainage from the depth of the cavity.

Dr. Lewis H. Taylor, Wilkesbarre, Pa.: The unfortunate cases are as instructive as the successful ones. I was called in consultation to see a patient, a youth, aged 17, complaining of eye trouble, who had been injured by falling from a bicycle. Examination showed choked disk. I thought it was either albuminuric retinitis, or possibly brain abscess or tumor. The first condition was ruled out by the urinalysis. The patient had intense pain on the right side of the head. An exploratory incision was made, trephining to find abscess but none was found. The wound was closed. Eventually the patient died after suffering intense pain in one side of the head. Necropsy showed a large abscess on the left side in the left temporal region. There were no ear symptoms at all, but the patient died from brain abscess on the side where there was no pain.

Dr. Crockett: I infer from what Dr. Lillie stated that his experience is the same as mine. I do not think the question of early operation is material. It is important to wait until the condition is well walled off. One need not be in a hurry about operating. Two of Dr. Cahill's patients were in a far advanced condition, practically moribund. Dr. Cahill spoke of one as an antemortem operation. There was double atrophic neuritis, complete aphasia, paralysis of both seventh nerves and of the sixth and the third on the affected side. Undoubtedly there had been an abscess for four months. It was an acute streptococcus abscess and the patient was dying when put on the operating table. A patient with hemianopsia, on whom an operation was performed by Dr. Jack during the war, who had temperosphenoidal abscess, and who passed out of observation for several years, came back to the infirmary in 1921. He had had epileptiform convulsions every two weeks for two years. He had hemianopsia, and a definite diagnosis was made. The patient went away for two years, so that he had temperosphenoidal abscess for four years before he was operated on, and then he finally made a perfect recovery. I think that one of the most important points is not to operate until you are certain what you are going to do. In the patients who recover, the localizing diagnosis is made before the skull is opened, when there is minimal laceration of the brain. This drain looks murderous but it does not lacerate the brain at all. I think the Poynting encephaloscope does not lacerate the brain. The drain can be allowed to stay in for six weeks. We had to do another operation to get the drain out, but that was an intracerebellar abscess. The patient made a perfect recovery. In one point of technic I differ from Dr. Cahill. I do not use a trocar to find the brain abscess, as you may pass the abscess. We have opened all brain abscesses by using a long artery forceps, then opening the blades. The pus comes out well if you strike the abscess. There was a report by Koenig last week on the use of artery forceps in opening brain abscess, and then passing the Mosher drain through the jaws. He states that it is a much easier operation. Exploratory operations for brain abscess should not be done. The aftertreatment is

materially shortened by the use of the Mosher drain. We can certainly get a 50 per cent. recovery rate from its use.

Dr. Ewing W. Day, Pittsburgh: My impression is that this report and also that of Dr. King, emphasize an important point: There is something radically wrong with the technic we formerly used in brain abscess. Dr. Cahill quotes 100 per cent. results. Dr. Mackewen quoted 100 per cent. results in the past, but Dr. Dench has had the courage to speak out and say that we have all taken this report with a grain of salt. If we take all the brain abscesses that have been operated on, the high tide for recoveries would be 10 per cent. of the published statistics. There were from 75 to 80 per cent. failures. If that is the case, there is something wrong in the technic. I believe the great fault, and one which Dr. Mosher has overcome by the use of the drain, is that the abscess having been found is lost again before the drain is inserted. The drain is inserted in the tissue on the outside of the cavity, opening new fields of infection and causing additional trauma and ground for development of encephalitis. Early operations are not as successful as those performed later. There is early encephalitis with more infiltration, but later, when a capsule has formed and the edema has somewhat subsided the prospects of success are better. I congratulate Dr. Cahill on his series of cases, but if the drain is adopted throughout the country the mortality will be very high. The results lie more in the technic than in any brilliant instrument for drainage.

Dr. D. E. S. Wishart, Toronto: In our recent cases we have used the Mosher drain; it is not of the type shown here but the best modification of it that we could get. We have had two cases: One patient died; the other recovered. We have had another extremely interesting case recently. A child was sent in by a pediatrician who made a diagnosis of meningitis or brain abscess. The child had had otitis media four weeks previously. There was no further trouble in the ear. The hearing was good. The diagnosis was made on intermittent vomiting and stupor. There was no nystagmus. The cell count was 1,050. There was no Kernig or neck sign. I made a labyrinth test on the child and response was normal on both sides, with this exception, that at the conclusion of each caloric test there was conjugate deviation of the eyes on the side of the slow component. The diagnosis puzzled everyone. The child died suddenly and a necropsy was performed. The middle ear was perfectly normal. There was practically healed lateral sinus thrombosis. There were two brain abscesses, almost symmetrical on opposite sides of the temperosphenoidal lobe. The child died from rupture of one into the lateral ventricle. This case illustrates the difficulties encountered in the diagnosis of brain abscess.

Dr. Smith: I quite agree that early operation is of no help in the average case, but is a disadvantage. We should take time to examine the patient thoroughly, with all possible tests, and seek the proper localization of the pus formation; then it is simple to operate. Another important point is to leave the drain in place for a sufficient length of time to ensure a more or less permanent opening, which eventually will close up. I was interested as to the site of operation in the cases quoted. My patients were operated on through the avenue of infection in which the gross lesion was found. In the winter of 1924, there was a meeting of the Section of the Academy of Medicine of New York and the College of Physicians. There was at Philadelphia a discussion on cases presenting severe symptoms in which the abscess was located and properly drained. The results showed that most patients

were well twelve and fourteen years afterward. I endeavored to use the Mosher drain in two patients, but was not successful. I think I must have used the type that he had discarded. In future, I shall use the medium sized one. I have not any reason for thinking that in the fatal cases the abscess was not well drained. Certain patients are apparently on the road to recovery, when they die suddenly. I do not know the cause of the sudden death. Dr. Fisher said the Bárány reaction should be employed whenever possible. That is absolutely true, but in many cases this cannot be done because the patient is in too bad a condition. We have to operate before we can carry out any tests. For many years, it has been our custom to use an instrument to evacuate the pus. This is not an artery forceps, but we call it the Jackson forceps. I do not know whether it is Dr. Jackson's invention or not. The use is similar to that of the artery forceps, and when we find the pus we evacuate it and introduce the drain.

Dr. Cahill: I should like to emphasize one point. About 60 per cent. of these cases show something through the mastoid route. I think this drain is very valuable in acute cases of abscess without a wall, it gives an opportunity for the wall to develop. Removal of the drain is difficult; so far, we have not found a means of removing it easily. We allow the drain to be gradually expelled by the brain. As to the value of doing lumbar puncture before opening the brain, to reduce the pressure to 250 mm. with the water manometer, it is important to consider whether that is going to help or injure the patients. It may prevent the secretion from traveling along the stream. Patients sitting up have tremendous decrease in ventricular pressure, and to remove fluid from the ventricle in this position we must use suction. I have not tried draining with the patient sitting up. There is danger of herniation. Will lumbar puncture cause localized meningitis to spread or not? The vessels in the white matter run perpendicularly, therefore when the infection is in the cortex, it will run along the vessels. I think too much consideration has been given to early diagnosis and to technic. I operated on six, and six patients were operated on by other men. We all used the same methods and we got the same results. I believe we must ascribe the favorable results to the technic of the drainage.

Dr. Day: Were all of the operators men from the same institution?

Dr. Cahill: Yes, but the results from the use of the drain are the same in other hospitals.

Some Remarks on Labyrinthine Surgery. Presented by Dr. Dench.

The cases are divided into first, those of localized labyrinthitis without symptoms. There is simple exposure of the diseased area, and the labyrinth is left alone.

Second, there is invasion of the labyrinth accompanied by symptoms. These call for drainage either by the Hinsberg or by the Neumann operation, preferably by the former. Seven of these cases are reported. All the patients recovered.

Third, cases in which the labyrinthine suppuration spreads to the meninges. These patients were all operated on by the Neumann method with drainage of the subdural space. All except one died.

Accidental removal of the stapes, per se, does not necessarily call for operative interference. Packing off the region of the stapes by separate packing is ordinarily sufficient.

The presence of a dead labyrinth without symptoms does not necessarily call for labyrinthectomy at the time of a radical operation. The radical operation may be performed and the dead labyrinth left in situ without harm.

Extensive sequestrums involving the labyrinth are not infrequently found in cases of chronic middle ear suppuration. Few labyrinthine symptoms may be present at the time of operation, although the bony destruction may be extensive. The patients usually recover after operation.

DISCUSSION

DR. KERRISON: I think Dr. Dench's paper is particularly interesting, because he deals almost entirely with questions on which there is a great diversity of opinion. Let me for the purpose of clarity state these questions: 1. Shall we, in the acute stage of suppurative labyrinthitis, immediately drain the labyrinth for the purpose of forestalling meningeal infection, or shall we leave the labyrinth alone, hoping first to bring the patient through the critical stage? 2. When confronted with a case in which a radical mastoid operation is required, in which there is a dead labyrinth, shall we do a radical operation alone, without draining the labyrinth? 3. Following surgical or accidental removal of the stapes, shall we drain the labyrinth or avoid operating on the labyrinth in order that the vestibule shall not be unprotected? Dr. Dench says that in the acute stage of suppurative labyrinthitis, characterized by vomiting, vertigo, nystagmus to the opposite side, he performs an early operation. In the case of dead labyrinth, with no symptoms beyond deafness and loss of vestibular reaction he believes that when any operation is necessary radical operation should be performed and the labyrinth should be left alone. There are many distinguished surgeons who hold the same view. There are other good surgeons who do not altogether accept these views. It may, therefore, be interesting to state that most men are timid on one point or another, but there are differences of opinion on the amount of danger in the points brought out. My experience on these cases convinces me strongly of two factors: 1. In the acute stage, operation is attended by maximal risk of meningeal infection. 2. Later, in the chronic stage of labyrinthitis, the dangers of labyrinthine operation are relatively negligible. Physicians who hold this view will not accept all Dr. Dench's indications for operation. Some surgeons feel that the best treatment during the acute stage is to avoid all surgery not absolutely demanded by the conditions present and keep the patient quiet until symptoms of acute vestibular irritation have passed. The opinion of many workers is that it is better not to operate during the acute stage. I cannot say that such a method would result in recovery of all patients but the dangers of the operation at that stage might do away with the advantages. As to the dangers of operating during the chronic stage, shall we do the radical operation without draining the labyrinth? I believe the majority of surgeons agree with Dr. Dench. My own feeling is that it is safer to drain the labyrinth. My belief is based on experience. In cases in which the radical operation alone has been performed the patient died of meningitis. I am convinced also that, in the chronic stage, the danger of opening the labyrinth is slight. My experience of surgery of the labyrinth is not very large. I have operated on seven patients with chronic suppuration of the middle ear complicated by dead labyrinth. All of these recovered. On the other hand, one patient, a case published years ago, on whom I performed simply a radical mastoid operation, and omitted to drain the dead labyrinth, died subsequently of suppurative meningitis. Dr. E. P. Fowler, at

the fall meeting of the New York Otological Society, reported a case of dead labyrinth in the chronic stage. He performed a radical operation and not finding any gross defects, did not drain the labyrinth. He brought this case forward for discussion. In February he reported that the patient had died of meningitis. Dr. Scruton told me that in another case of meningitis, after the acute symptoms had passed a radical mastoid operation was done and the labyrinth was not opened, and the patient also died of meningitis. Some years ago Dr. Wendell Phillips reported, at the New York Academy of Medicine, a case in which he had performed a radical mastoid operation without opening the labyrinth. His patient also died after meningitis had supervened. Therefore, shall we perform radical operation and leave the labyrinth alone, or include the labyrinth in drainage? This cannot be decided on the many cases in which radical operation alone results in recovery. There are a small, but not negligible percentage of cases in which such procedure results in meningitis and death. Dr. Fowler's and my operation had this result.

Another question is that of fistulas. Operators make very careful search for fistulas in the semicircular region or maxillary process behind the path of infection. If we do not find it, and if we find an intact labyrinth, I do not think this should be considered a contraindication for not draining the labyrinth. I think in my case and in Dr. Fowler's that it was absence of fistula that decided us — yet the patients died.

In regard to the views expressed on accidental removal of the stapes during surgical operation. Some of these patients recover, but without question it is a very serious condition. The old statistics of Youngster in nineteen cases of accidental removal of the stapes, still hold good. In thirteen of these cases the labyrinth was drained, and nine patients recovered. That left six patients not operated on, of whom only two recovered. Two years ago, Dr. Richards reported cases in which during radical operation the stapes was removed. The patients died rapidly of meningitis. This accident also occurred in the hands of a hospital surgeon, and that case also resulted in rapidly fatal meningitis. I think both of these patients could have been saved if the labyrinth had been promptly drained. I have not had any experience with that accident because I am so timid that I leave the granulations in the notch of the oval window alone rather than take any risks. If we have a bulging drum we might make an incision here and we might bring out the stapes. We try to avoid infection but there may be pus down as far as the ganglions. When we have infected the tympanum we make an incision, but in the other case, the vestibule is full of pus which may cause death. Why should we not make an incision and use a little pressure to evacuate the pus. If the ear becomes infected, drainage is better and the meningeal infection will be reduced. Dr. Dench's figures of stapes extraction with five recoveries are gratifying, but the results must be ascribed to his skill in the first place, and, secondly, to good fortune. The results in acute labyrinthitis are unusual, and I offer my congratulations.

Dr. John B. Rae, New York: Some years ago, a physician read a paper on this subject and said that if in a case with one dead labyrinth we find on rotation that the after nystagmus is equal on both sides this is proof of a healed dead labyrinth. I do not know if that is true; if it is true it is a positive help, but it justifies the radical operation rather than a radical and labyrinth operation in the presence of a dead labyrinth. I would like to know if Dr. Dench agrees.

Dr. Tobey: I have two cases of radical operation with dead labyrinth, of many years' duration. Dr. Dench makes me feel I am safe. Dr. Kerrison makes me feel I am not safe because his patient died. I took the consensus of opinion of the hospital staff at the time and we decided to leave the labyrinth alone. Apparently Dr. Dench has had no difficulty.

Dr. M. A. Goldstein, St. Louis: Dr. Dench has given us safe and substantial points of view. There is one suggestion which has occurred to me on several occasions and I offer it for what it is worth. There may be a practical value in some of our investigations which will help our surgical judgment. I use the big tuning fork before a surgical operation. Assuming that the labyrinth is healthy, at the time of radical operation, the tuning fork will lateralize to the same ear. If the labyrinth is involved the tuning fork will lateralize to the opposite ear. This will give you an index of labyrinthine involvement even before you find nystagmus.

Dr. Edmund P. Fowler, New York: That reminds me of a diabetic patient I treated some years ago. The labyrinth was dead, but the forks referred to the dead side. The labyrinth came out as a sequestrum. The patient was operated on and after operation the forks still referred to the same side.

Dr. Kerrison: Some years ago, Bárány reported in a radical operation that all the hearing tests showed absolute deafness except the Weber test. He explained this on the grounds that there was a large cavity acting as a sounding chamber, which gave the patient the impression that the sound was heard in that ear.

Dr. Lillie: Another problem which we have encountered is this: In acute otitis media, surgical mastoiditis with meningitis and seventh and eighth nerve neuritis, are these cases due to choked seventh and eighth nerve, to involvement of the vestibular and labyrinthine ganglions, to the end-organ or to dehiscence in the middle ear? In one patient I performed an operation and found nothing in the labyrinth, but found localized meningitis at the apex. I took care of this man by Dr. Dench's method, with good results. In a case of another type, in which there was an embolic labyrinth following typhoid vaccine, the labyrinth ganglions were out of commission, but there was no involvement at all. I would like to hear some opinions on the treatment of such a condition.

Dr. Norval H. Pierce, Chicago: Regarding the last two cases reported, labyrinth operation would be contraindicated. I believe these are cases of radical axis, not due to any suppuration, although total deafness might ensue after injections of any serums, with complete suppression of the static apparatus. These patients almost always recover later. I have had two interesting cases of this condition, one occurring in a plethoric man, who came to my office suffering from almost total deafness, but without any marked dizziness at any time. The onset of this deafness had occurred within twenty-four hours. It had grown markedly worse during that time, and when I saw him he was almost totally deaf. The turning and the caloric tests elicited no response. We thought of syphilis, but this was denied. We made a spinal fluid examination with negative results. I was puzzled. I questioned him about quinin and other drugs. He remembered that while on board his yacht he was struck with a wad of gun cotton, and a physician on board, fearing tetanus, injected him with antitetanus serum. That may seem long drawn but we know that there is a delayed anaphylaxis in these cases. Another case was that of a man from Oklahoma, who had partially recovered from otitis media.

He gave a history of sudden deafness occurring in the other ear. When he came to the office he was very deaf. He gave a history of having had antidiphtheritic antitoxin some months before. When he left my care there was distinct improvement in hearing in both ears. The hearing distance for whisper was 24 inches. The other ear improved with liberation of the pus.

These anaphylactic cases do not call for operation.

DR. LILLIE: I did not have reference to patients injected with any serum. In experimental animals, such as rabbits and guinea-pigs, there are sometimes labyrinth symptoms. We find pus in the labyrinth. I would like to know whether the members think there can be suppuration in the labyrinth without otitis.

DR. FOWLER: One point should be stated. The presence of pus in the middle ear does not mean that labyrinthitis is present. We continually see patients in the large clinics in whom deafness suddenly comes on with labyrinth symptoms, with no signs whatever of any pus. There is another reason why there may not be mastoiditis and a dead labyrinth coincidentally. We cannot tell the difference, but there are fewer deaths from labyrinth surgery because we are operating on fewer patients.

DR. DAY: There is a change in point of view as to treatment, and the change is toward conservatism. The patients on whom operation was performed died, but we cannot prove that they would not have died if no operation had been performed, as the infection passed from the labyrinth to the meninges. We have mentioned guidance by tests. If the spinal fluid pressure is markedly increased, I take it that the labyrinth is involved and that the infection has passed to the meninges. If the fluid pressure exceeds 100 mg., the infection has reached the arachnoid space. However, any infection will make some difference in pressure on account of absorption of toxins. It is easy for infection to reach the labyrinth if the pathways are not blocked.

DR. SMITH: I agree with Dr. Dench that in the case of fistula of the horizontal canal, discovered during operation, and also in case of dead labyrinth which does not give symptoms, it is best to leave these things alone. In acute cases of mastoiditis, sometimes we have marked symptoms of labyrinth involvement, but sometimes these will disappear after a simple mastoid operation. If meningitis supervenes, I agree with Dr. Dench and Dr. Day that the patient will die in any event. You get the experience of the labyrinth operation, but that is all. Chronic labyrinthitis is another question. If there are very definite symptoms which point to operation it is justifiable to operate, but if there are few symptoms, operation can be left for future consideration.

DR. RANDALL: The whole subject under discussion is surgery of the labyrinth. I was trained under Norris, who taught us to go to the eye dispensary to study labyrinthine findings. I should like to say a word as to the medical aspect of this matter. In spite of the severe anaphylactic shock which struck one of our members when Dr. Theobald spoke of mercurialization in some of these cases, I have noticed gratifying results following the use of Donovan's solution of arsenic and mercurial iodid. I have hesitated in using surgical intervention. The matter of response to medication has been carefully tested. It is a matter which deserves attention in a surgical subject.

DR. TOBEY: I would like to disillusion Dr. Day in regard to the dynamics of the cerebrospinal fluid. There is no relation between meningitis and the pressure of the fluid.

Dr. Arthur B. Duell, New York: I have passed from the enthusiasm of operating and have grown somewhat conservative in my management, although there are conditions in which the labyrinth should be drained.

Dr. Dench: As I understood Dr. Kerrison, he would hesitate to open an acute labyrinth for fear of doing more damage. He would wait for it to become walled off. I lost two patients in that way. I do not think there is possibility for erosion through the semicircular canals, but with *Streptococcus mucosus capsulatus* infection, which is a very dangerous organism, I was afraid that if I took out the labyrinth there might be extension by the cerebrospinal fluid. The man would have died by the surgeon instead of by the disease. I do not say it is dangerous to take out a dead labyrinth, but that it may not be necessary. In many cases the labyrinth is dead and can be left alone. After a mastoid operation, there is no need to increase the time and danger of an operation. I am one of the timid ones, and consider a labyrinth operation one of the most difficult. In regard to removing dead labyrinths, we do not know that they have been destroyed, we do not open unless we have to. I do not want to have to drain the labyrinth every day I perform a mastoidectomy. The first time I took out the stapes accidentally I had a sleepless night. Now it does not bother me. The patients do not die. I believe we test the interior of the labyrinth by rotation tests, but it cannot be proved. If you have extirpated the labyrinth, the rotation time is equal (50/50); there is complete obliteration of the semicircular canals. With reference to the lateralization of the tuning fork mentioned by Drs. Fowler and Goldstein, I do not know whether it happens in all cases, but it does in some in which there is labyrinth destruction from suppuration. I think it is through the nerve that we get this interpretation of sound. We do not know about interpretation of sound through the auditory nerve, whether these vibrations are not mistaken by the patient and interpreted as sound. In regard to Dr. Smith's remarks, the labyrinth symptoms would disappear after a complete mastoid operation. I think there was a perilabyrinthitis. In one case there was chronic suppuration on one side and a dead labyrinth on the other. The patient was absolutely deaf with vertigo. We performed a radical operation for the suppuration. In that patient it was due to inflammation about the labyrinth. The suppuration disappeared. I took a chance because I did not want to make the man absolutely deaf.

Dr. F. L. Jack, Boston: In connection with infection following removal of the stapes. In 1892, the first stapedectomy was done in a case of chronic suppurative otitis. When the ossicles and middle ear were cleared out the head of the stapes was seen to be carious. We decided to remove the stapes. There were no symptoms following that. I had ten cases without any infection but it was pure luck, I would not want to do that now.

Afternoon Session

Neuro-Otologic Examination in Seven Consecutive Verified Cases of Brain Tumor in the Posterior Fossa in Children. Presented by Dr. Wishart.

Seven cases of verified brain tumor in the posterior fossa in children are reported. Four of these were definite intracerebellar tumors. Three were extracerebellar but in the midline. Three of the patients had precisely the same lesion. Complete histories and neuro-otologic details are given regarding each case. The data have been summarized in four tables to permit easy reference. Photographs of the brain tumors accompany the paper.

In view of the information gathered from this and from the previous series of cases, I would now submit that our evidence permits the following postulates:

1. Tumors of the frontal lobes do not interfere with cochlear or vestibular function.

2. Subtentorial tumors which are completely outside the brain stem and the cerebellum affect homolateral cochlear and vestibular function.

3. Subtentorial tumors which are in the midline and which do not involve cerebellar tissue do not affect cochlear function. They do not interfere with vestibular function. The interference tends to be bilaterally symmetrical and the functions of both vertical canal reflex arcs are affected before those of either horizontal canal.

4. Unilateral intracerebellar tumors do not interfere with cochlear functin. They do interfere with vestibular function. The function of the reflex arcs of the vertical canals will both be affected before those of the horizontal canals. The tumor will be found on the side opposite the vertical canals with whose function there is most interference.

THE PRESENT STATUS OF VESTIBULAR TESTS IN INTRACRANIAL CONDITIONS. Presented by DR. FISHER.

The records of 103 suspected cases of brain lesion, seen at the University Hospital, were examined and classified. Those selected were verified by operation or by necropsy so that the diagnostic value of vestibular tests in such cases could be definitely estimated. The tabulation is as follows:

	Vestibular Examination Correct	Incorrect
Cerebellopontile angle tumor, 29 cases	27	2
Tumors of fourth ventricle, 3 cases	3	0
Midline infratentorial tumors, 2 cases	2	0
Cerebellar lesion, 28 cases	21	5
(Cerebellum ruled out by vestibular examination, 2 cases)		
Cerebral lesions, 28 cases		
(*a*) Hemispheric lesions	15 (correct side)	
Vestibular examination ruled out, posterior fossa	10	
Diagnosis made by vestibular examination alone	3	
(*b*) Pituitary lesions, 13 cases	10 (correct location)	incorrect location
Vestibulary examination ruled out, posterior fossa.	2	

Otologists are particularly interested in cerebellopontile angle tumors, because they are first consulted in these cases. The phenomenon complex of a totally destroyed ear on one side with nonresponsive vertical semicircular canals on the side opposite the lesion, and good responses from the horizontal canal on that side was found to be reliable with few variations.

Vertigo and past-pointing were reliable as to condition of posterior fossa contents. No definite phenomenon complex was found for cerebral lesions, but if the patient had signs of brain lesion and was easily made sick by turning and douching, the condition was cerebral. In conclusion, neuro-otologic tests conducted by otologists, reached the same conclusions as neurologic examinations, and in some cases the otologic examinations were the only method of study by which diagnosis could be made.

THE IMPORTANCE OF ANALYSIS OF THE COMPLETE VESTIBULAR RESPONSE. Presented by DR. J. Q. HOLSOPPLE, Baltimore.

In order to correlate nystagmus time with ordinary responses involving change in position, the phenomena of the semicircular canal mechanism were studied in a group of undergraduates in good physical condition, between the ages of 17 and 25. The postrotation nystagmus times for all reactors, both clock-wise and counter-clock-wise were measured, the variations being from nineteen to thirty-four seconds. A slow turning chair test, about a turn in eighteen seconds, was given, and showed wide variations in estimation of a complete turn, in blindfolded students. A test on the campus was made with blindfolded students, headed for a tree at 262 feet distance. Wide deviations were noted. The results of these and repeated tests showed that all of the reactors had normal vestibular apparatus. Yet when deprived of visual aid they showed great tendency to deviation, either to right or to left, and they were not aware of deviations. It would seem, therefore, that the vestibular stimulation must be of low intensity and of near threshold value. Thus the determination of threshold value for different persons seems of practical importance. We should have a measure of the minimal intensity of rotational stimulation required to provoke the vestibular response. These experiments should be extended to cover responses to rotation around other axes of the body, since canals other than the horizontal may affect orientation. The tests should be repeated until their normal variability is established and pathologic reactors should be compared with normal reactors in order to determine what extravestibular factors may affect the response.

In conclusion, nystagmus time is not the only, nor the most striking of vestibular responses.

DISCUSSION

DR. J. GORDON WILSON, Chicago: The most valuable thing about these papers is the different way in which the men have looked at the problems, psychologically and perhaps clinically.

An important point which has been brought out is that of the threshold of stimulation. I have a clear idea that the threshold is essential in the estimation of all ear tests. We have managed to establish the threshold of hearing, but we have not got the threshold of stimulation. With all due deference to the physiologists I think the perception of the threshold is very important. We can tell to a certain extent that the threshold lies near a particular point, and if we can get some method of estimating that readily, it would be an important step in advance.

With regard to the question of flying, there is doubt in regard to the movements.

We have had some interesting cases presented in the clinical papers. There are two points to consider in relation to this, first, the accuracy of observation, and I think the data presented both by Dr. Fisher and Dr. Wishart are of such nature that we can rely on their accuracy. When there is dubiety about a case we have to go back to the most accurate and reliable data obtainable.

When it comes to a question of deductions, we have some doubt in the question of douching the ear. We can get a difference of the two sides from pressure lesions, but when you deduce different pathways you are against a different proposition. There is a difference between effects and deductions from effects. Have we any evidence to show that we have separation of the verticals to the homolateral or to the contralateral side? I have found frequently that I get no reaction in the vertical canal, then I tilt the head back and get a reaction in the horizontal; I tilt the head back again, then I can obtain the vertical

response. That to me demands further explanation. We have evidence to show that the pathway from the horizontal canal is much more easily affected or traversed than the pathway from the other. The same stimulus, which will affect the horizontal canal will have no effect on the vertical. What does that mean? The reaction in the horizontal goes on after the vertical, but once the pathway is traversed or opened up it is much more easily traversed again, and needs much lower stimulus.

In regard to the question of putting down details, when Dr. Fisher talks about spontaneous nystagmus in all directions, what does he mean? I have seen tumor in the cerebral hemispheres, in the descending horn, give a spontaneous nystagmus in all directions. It was slow and quick. It was a sort of optical phenomenon. Does Dr. Fisher mean it was slow to the midline, or to a particular point? These details will help us when we come to settle the question of the function of the different parts. My experience is that there are very few facts in neurologic data that are of use in localizing a lesion. In our present knowledge the vestibule is a very valuable adjunct, but it must be tested by the man who gathers the details and harmonizes them with other details. The vestibular data and the oscillations of the eyes are suggestive of positions of the lesions. For instance, I find that in the cerebellar cases in connection with the oscillations of the eyes, we get other signs, such as tremor and swaying. That is indicative of cerebellar involvement.

Fifteen years ago, I reported experiments on animals in which we always found in lesions above the mesencephalon, a slow deviation of the eyes to the side of the lesion. That has been contradicted, but I know it is true. If there is a lesion above the mesencephalon, the caloric test makes the eye go to the side of the slow nystagmus, and indicates a lesion of the part which lies above the right nucleus. The caloric test is most important. In regard to the rotation test, I do not think it is of much value as regards clinical results. What we want is a picture not after rotation, but during rotation of the nystagmus. I know that there are different workers trying to construct apparatus to get that kind of evidence. A simple lesion of the cerebellar cortex can be localized by cerebellar movements. Constant disorders of pointing are very suggestive of posterior fossa lesion.

We are obliged to Dr. Wishart for saying that he has modified his views expressed in a paper given two years ago. If he will give another paper in two years from now, he will modify his present statements. He states that "tumors of the frontal lobe do not interfere with vestibular and ocular function." I am not sure about that because the cerebellum and the frontal lobe have very close communication. I, like Dr. Wishart, have made diagnoses of tumor on the wrong side. The question of rotation in cerebellar lesions is opposed to that seen in cerebral lesions. There is little toleration for vestibular tests. He speaks of nausea and vomiting—the nystagmus also is extremely violent.

DR. ROBERT SONNENSCHEIN, Chicago: In some cases when you give the stimulation with the head in the erect position, no reaction is noticed, even if the ear is douched for sixty seconds, but when we douche for three or four minutes, then retract the head, there is a horizontal nystagmus. If you then turn the head forward you get a vertical nystagmus. I wish that Dr. Fisher would give us the explanation of the phenomenon. Is it perhaps that the original douching has allowed, through cooling of the labyrinth, a reaction of certain pathways which were not susceptible to the reaction as normally present.

With regard to Dr. Holsopple and the threshold of minimal stimulation, I believe it is known to most of you that Kocher of Berlin devised a caloric

reaction by means of small quantities of water. In Alexander's clinic they used tap water at 60 F. and injected 5 c.c. into the ear. Then they waited from thirty to sixty seconds and if there was no reaction they used from 10 to 20 c.c., then increased the quantity. In the vast majority of cases they got minimal reactions with 10 c.c. It does not produce nausea and vomiting, but vertigo and past-pointing are produced.

DR. DENCH: I have had one experience which controverts the statement that tumors above the tentorium show no evidence on stimulation of the semicircular canals. I made a diagnosis of subtentorial tumor from the variations in the caloric test on that side. I think I obtained a vestibular response. In regard to the statement that there is a cerebellar response without the cerebral response, in this case the response to the nucleus of the eighth nerve or cerebral or quick component was entirely lacking. Therefore one cannot make a diagnosis on one test.

DR. FOWLER: Dr. Holsopple's paper interested me in several points. When we think of the tremendous patience on the examiners' part and the patience necessary to elucidate these tests, there must be something wrong. I think that if we had had a lot of children and started them for that tree with a prize at the end of the game they would have got better results than those students did. Sometimes students' minds are not focused on the subject. It seems to me that the variations in reaching the tree were too wide. When we blindfold patients in our tests, they can walk straight down a 50 foot room. This depends on the speed. If they walk rapidly they are able to go in a straight line. The speed with which the patient turns, stops and starts has tremendous influence. All these points might not have been covered, because Dr. Holsopple does not give us the clinical side.

DR. DENCH: With reference to Dr. Fisher's paper, since the foundation of the Neurological Institute I have been consulting otoneurologist. For the last three years the work has been taken care of by Dr. Sauer. The work is now being carried on more thoroughly, many more experiments have been made and there are fewer errors in the otoneurologic diagnoses, as compared with the diagnoses of operating surgeons. I think we should cooperate, as Dr. Fisher has suggested. The routine otoneurologic examination is of great importance when an intracranial lesion is suspected.

DR. WISHART: Dr. Fisher gave instances of several supratentorial tumors in which the findings were different from mine. In regard to Dr. Wilson's remark, I may recast my statements, but so far evidence tends to confirm my deductions. I would like to know how Dr. Fisher made his diagnosis of cerebral tumor in the case of absence of rotary nystagmus after the caloric tests. From the evidence he gave I would not have reached the conclusion he did.

Dr. Wilson has noted that on a number of occasions he has observed no rotary nystagmus in the patients in the upright position. If he puts the patients back horizontally, and then puts them upright again, he gets vertical nystagmus. I have observed the same phenomenon, but when you look at case histories reported today there are several cases in which you have recorded as a distinct observation that there was no rotary nystagmus with the head upright. The head is then put horizontal then upright again and again there is no rotary nystagmus. There are probably two groups of cases, one in which there is no rotary nystagmus in the vertical position, and one in which there is rotary nystagmus after the horizontal nystagmus. As to the rotation tests, I have given up rotation for children, it is of such limited value. I use it only in a small number of cases.

DR. FISHER: The vestibular reaction does not depend on one particular type of test or response. Vestibular reaction presents a complete picture, and the experienced examiner has learned how much value to place on certain things. To state that the nystagmus is 20 or 30 in duration will not tell the entire story. There are certain things that examiners can learn by studying the nystagmus and these will tell a story to him which is not described in textbooks. We try to avoid generalization statements and to work out the complex formulated groups of findings to fit certain types of cases. We have formed a phenomenon complex for cerebellopontile angle tumor, but we cannot depend on written description and we cannot describe it in words. The vestibular examination in its entirety is only a part of the picture. It should be studied by the physician in charge of the case. In the cerebellopontile angle tumor, the etiologic picture is sufficiently plain for diagnosis. In other locations the vestibular reactions may suggest things to complete the rest of the diagnosis, as in cerebral or pituitary lesions.

Dr. Wilson spoke of spontaneous nystagmus; that meant vestibular nystagmus. If it is optic nystagmus we call it optic nystagmus. When it is not, we call it vestibular. The same movement is quicker in one direction than the other. When it is evident in every direction the patient looks in those directions. When he looks ahead and there is nystagmus, it is unusual, and we make a note of it.

In regard to rotation not being important, the caloric test is the most refined test because we can examine one ear or one portion of the ear, but for the purposes of clinical diagnosis the rotation test is most valuable. Our knowledge of the integrity of the cerebellum depends largely on whether we can test vertigo and past-pointing, and the rotation test is the best for that. Patients with a normal cerebellum will not past-point after douching, but will past-point after rotation. Whether you get double stimulation, I do not know. Past-pointing is more readily elucidated after turning than after douching. Frontal lobe tumors, I have found, do cause loss of response from the perpendicular semicircular canals and will markedly affect the eye response and give evidence of irritation of the vestibular mechanism. All of us have noticed the phenomenon of retarded nystagmus, appearing only after the second change of position. However, I have noticed that such retarded nystagmus will not last very long, with few exceptions. We douche the ear four minutes before we conclude that the vertical canals will not respond. After four minutes the vertical response will be of short duration. I think it is evidence of definite impairment of some sort. This finding, together with another finding, might be very valuable.

In regard to minimal threshold stimulus, all our tests are stimulation tests. In the rotation tests we attempt to measure the duration of the response and the quantity of response that follows this test. In the caloric test we test the threshold of stimulation, and how long it takes to chill the ear. I do not think the quantity of fluid you inject means much. It is keeping the ear chilled all the time you are testing it, no matter what the size of the stream. It is surprising to me how much Dr. Wishart can do with children in the neuro-otologic work. I think children are most difficult to test. The rotation test is almost useless. If you do not elicit past-pointing you cannot conclude that it is absent.

DR. HOLSOPPLE: I tried for two weeks to have the subjects react on the straight line. Indoors, all reactors walked straight for 40 feet. When out of doors they lost the echoes from the walls and ceiling. They deviated at the start, which in 100 feet would take them away out of the quadrangle. The sense of direction is quite different in the open air.

DYNAMIC STUDIES ON THE CEREBROSPINAL FLUID IN THE DIFFERENTIAL DIAGNOSIS OF LATERAL SINUS THROMBOSIS. Presented by DRS. TOBEY, JR., and JAMES B. AYER, Boston.

This study is based on two important statements: Increased intracranial pressure is normally propagated throughout the cerebrospinal fluid system and may be measured by a manometer employed in connection with lumbar puncture. Compression of the internal jugular vein causes increased intracranial pressure with immediate rise in spinal fluid pressure.

Absence of rise in fluid pressure (positive Queckenstedt sign) indicates spinal subarachnoid block (as in spinal cord or cerebellar fossa tumor). Further extension of the use of this test is to detect evidence of block by lateral sinus thrombosis, partial or complete, thrombosis of the jugular bulb or internal jugular vein. The mechanism in venous obstruction differs from that of tumor, in that there is no elevation of pressure at its source, because of venous obliteration. The test for sinus thrombosis includes the examination of pressure effects on each jugular vein separately, and not with simultaneous compression of both veins as in the diagnosis of cord tumor.

In a typical case of lateral sinus thrombosis there is a prompt and rapid rise in fluid pressure to twice or three times the initial reading, on the normal side, this pressure rise being maximal and equivalent to the pressure attained when both jugular veins are compressed. Pressure over the thrombosed lateral sinus causes either no rise, or a slow rise of only from 10 to 20 mm. in the manometer. Partial obstruction gives less striking results, yet is of value as corroborative evidence.

General septicemia is not a contraindication to the test.

The test is of special value in the presence of double mastoiditis developing symptoms of lateral sinus thrombosis.

DISCUSSION

DR. AYER: It has been a great pleasure to be associated with Dr. Tobey in this work, which has developed as we anticipated it would, although there have been many minor problems which have had to be worked out in doubtful cases of mural thromboses.

In the physiologic demonstration of the circulation of the brain we have used the cerebrospinal fluid as indicator. Before speaking of the test as we used it in connection with lateral sinus thrombosis cases, it would be clearer if I gave you the development of these dynamic tests in other conditions. The value of the test was brought out first in 1916 when it was shown that compressing the jugular veins causing temporary block might increase the intracranial pressure due to cerebral stasis, which was propagated to the cerebrospinal fluid. This could be checked up by measuring the cerebrospinal fluid pressure. The manometer is attached to a part of the arachnoid space or any part of the ventriculo-arachnoid system and the rise in fluid pressure measured. This is applied to cases of cord tumor or other blocking of the spinal subarachnoid space.

If the tumor compresses any part of the spinal subarachnoid space, compression of the jugular veins in the neck will no longer cause rise in pressure, registered at the point of the manometer. There is no change when there is pressure at any other point. This is clear when we use two manometers. The second manometer is inserted at the cisterna magna, but when the veins in the neck are compressed, the cistern pressure rises, and the lumbar level remains the same—normal above and abnormal below the block.

Recently this work has been extended and now we have a series of cases in which the tumor is in the cerebellar fossa. We then put the needle in the lumbar sac, and also a needle in the ventricle. The pressures will be the same to start with. Compression of the jugular vein will cause rise in the ventricular needle, and no rise, or very slow rise in the lumbar needle. The rise is not so high in the ventricle, showing pressure block from the tumor in the cerebellar fossa.

The basis on which this present work depends is that in pressing the jugular veins on both sides one causes venous stasis. We may repeat this test at different stages, indicating that the pressure is direct into the ventricular system. The fluid rises in the ventricular system and is propagated downward until it strikes the block, beyond which it cannot pass.

Clinically, we hope to apply this to the diagnosis of sinus thrombosis. If, in the presence of either jugular thrombosis or sinus thrombosis, we compress the jugular vein there is cerebrovenous congestion. There is nothing shown in the lumbar manometer, but if we compress the other vein we cause excessive pressure which will run up high. Here our tests consist in producing no rise of the pathologic side but excessive rise on the other side. This sounds easy, and it is easy, but there are a number of factors which make the problem complicated.

During the work on tumors in which we have used the routine neurologic examinations, we note we get a very poor rise on pressure of both sides. We have no guide by another needle as we have with a tumor of the cord. These, however, almost always run a low spinal fluid pressure. If they have an aqueous pressure of 150 or more they will show a good rise. Three times we obtained unusual pressure on compressing both jugular veins. In persons with thick necks, until we are successful in finding the jugular veins we do not get a proper rise because of faulty technic.

A more important hazard is abnormal rise under pressure. If the jugular veins are not compressed with great care the patient will cough. He is strangled. This will immediately throw up his level of intracranial pressure which will give a false rise in the manometer. These faults are common in the neurologic clinic and also in this work. Another bad feature in connection with this work is the anatomic variation of the sinus veins and their tributaries. We have seen at one operation sinuses of varying size. I am told this is not unusual. This would cause varying pressure on the two sides, and this is a feature which we must take into consideration. If the compression is not accurately placed we have certain anastomotic supplies which will give a slight rise in cases of block, which can be interpreted as anatomic variation when the pressure does not come up on the affected side but comes up on the normal side. With a slight rise on one side and a moderate rise on the other, we must not jump to conclusions too quickly. As Dr. Tobey says, we have many more sinus thrombosis cases than are diagnosed.

We have several thousand cases to study. There are many in the neurological clinic. We have to study what is normal and what the difficulties are. With cord tumors the method has proved quite satisfactory. In the work on brain tumors, it is new, but so far satisfactory. In the work on thrombosis it is apparently dependable in connection with complete thrombosis. With a mural thrombus or partial thrombus, it is not so dependable.

DR. DAY: We have had a few cases and I think we can diagnose internal hydrocephalus by the absence or presence of the respiratory waves. In our cases we found positive results in internal hydrocephalus with blocking of the aqueduct of Sylvius. If the blocking is complete, there is absence of the respiratory wave; with a free aqueduct the respiratory wave is present. We have acted on that sign in a number of cases.

DR. RANDALL: In studying the eyegrounds with regard to findings in suspected intracranial cases, I obtained a more steady view of the eyegrounds if I raised the head of the patient and bent it forward. I did not realize why until I read the observations of a Mexican worker who showed that this caused dilatation of the pupil, which he used as a rule of thumb way of testing the matter, so that with the compression of the cistern and other parts of the upper cord he obtained a distinct amount of increased pressure reacting on the cranial contents and thence on the pupils. I am sure that this will be of material service to us. As to the question raised by Dr. Ayer on variation of the jugulars on the two sides, we sometimes find no internal jugular at all, the lateral sinus passes out at the external jugular and the mastoid emissary. Thus, pressure may be transferred through the vertebral veins and have influence through them, bringing in another aspect of the case.

DR. CROCKETT: If I had had three or four of Dr. Tobey's patients, I would not have operated on them in time. I do not think Dr. Tobey would with the clinical facilities we had at that time. I think there is an important differentiation in cases of double mastoiditis. That is very difficult, as I can recall in a case. The child had a typical septic temperature of lateral sinus thrombosis. The entire mastoid was tender. The only thing I could possibly do was to tie off the jugular on the side which looked the worst. Fortunately, I found the correct side. With Dr. Tobey's method we would know what to do and we would operate at an earlier stage. Undoubtedly, there are many patients with lateral sinus thrombosis who ultimately recover and never need operation.

DR. FOWLER: As I understood the speaker, in the partial clots you do not notice so much change in the sign. I think that will be the weak point of the method. For instance, I remember one patient with a positive blood culture, and also beginning erysipelas, in whom complete thrombosis of the vein occurred. I think it should be borne in mind that unless there is sufficient blocking this method is not enough.

DR. SMITH: This work gives us a great advance in the diagnosis of lateral sinus thrombosis. To repeat the experience that our President has had in finding which sinus is involved in double acute otitis media with mastoid involvement, when you know there is sinus involvement you don't know on which side to operate. I had a similar case. With blind luck I got the right sinus; I had one case in which I got the wrong sinus. I operated later on the other sinus, but the patient died. It shows the difficulty of choosing the right side. I had the assistance of two competent men. I wish to congratulate the readers of the papers on their contribution to the diagnosis of lateral sinus thrombosis.

DR. AYER: Dr. Day's suggestion in regard to hydrocephalus is interesting. I think that observation holds. There is another, possibly a better test. If you measure the amount of fluid taken against the pressure drop, the fluid is much more than in a normal condition. It is a quantitative test. I think that is more reliable than observations on the respiratory excursions. I would like to suggest to Dr. Randall that instead of being related to intracranial pressure, the phenomenon he mentions might be due to the ciliospinal reflex, which is the result of compression of the neck. Dr. Tobey mentioned something which I should have mentioned because of the extreme importance of the matter. In 1919, with a number of other men I cooperated in a paper in which we stated this biological principle that, given a cerebrovenous pressure leading to meningitis in the experimental animal, if you have organisms virulent for the meninges, the drawing off of pressure will facilitate passage of the organisms into the arachnoid. I am satisfied that this principle exists. This is laboratory work, and we have not

had enough proof clinically that it exists in man. While we approached this with temerity, we thought it was a serious matter. In a group of patients with known septicemia, we might precipitate meningitis. I believe it is biologic in animals, and probably in man, but we have to have enough organisms and the right organism. It might not happen once in a thousand times, and we should not stress it, but if we did precipitate meningitis, we should stop the test the next day.

There are no other fluid tests of importance today in regard to sinus thrombosis. There are few cells, sometimes increased pressure, sometimes increased proteins, sometimes diminished proteins, with a mild hydrocephalus. Therefore dynamic tests are the only ones of value in sinus thrombosis.

DR. DAY: Is there danger in the presence of bacteremia in meningitis, if you draw off a small quantity of fluid?

DR. AYER: Cats have about 34 cm. of fluid. In order to precipitate meningitis take away half the fluid and the pressure drops to zero. This is not the case in man, but with jugular compression you have prompt rise and both factors facilitate the passage of organisms.

DR. DAY: What is the normal amount in man?

DR. AYER: About 150 c.c., but it varies a great deal. I have never seen the result in man.

DR. DAY: With the manometer, do you draw off more than 2 c.c.?

DR. AYER: We take 10 c.c. and we lose a certain amount of fluid into the epidural space.

DR. RANDALL: What is the cause of opisthotonos in these cases? Why is the head drawn back, if it is not that the contrary position increases intracranial pressure?

DR. AYER: I do not mean that there was no localized meningitis. Your feeling was that cerebral pressure was increased by raising the head. I thought that the ciliospinal reflex might account for that.

DR. RANDALL: We have seen a patient pass into collapse by throwing the head back, on account of increase of intracranial pressure. We have felt that that was a serious objection.

DR. SMITH: Is it not true that the loss of cerebrospinal fluid is rather harmless in human beings? A few years ago they operated for relief of meningitis by excessive drainage of the cisterna magna. Dr. Dench did six such operations. It was surprising what an amount of fluid you could lose without detriment to the patient, much more than in this procedure.

DR. CROCKETT: Before the war, a physician in St. Louis treated meningitis by doing lumbar puncture with a permanent drain. He left no fluid in the patient. He reported two cures.

DR. TOBEY: In regard to making a definite diagnosis of mural thrombosis, in the presence of sinus infection I felt justified in making these tests. Some allowance must be made for my enthusiasm in trying to find these cases. We are justified when the conditions of pressure on the two sides point to it, in making a diagnosis of moderate thrombosis, and acting accordingly.

HERPES ZOSTER OTICUS: A CASE WITH INVOLVEMENT OF THE FIFTH, SEVENTH, EIGHTH AND NINTH NERVES WITH A COMPLETE VESTIBULAR EXAMINATION. Presented by DR. FRANK L. DENNIS, Colorado Springs.

This is a review of the pathologic studies of Herd and Campbell and Ramsey Hunt in herpes zoster, demonstrating the identity of the changes in the posterior

ganglionic chain in herpes zoster with those of the anterior horns in acute anterior poliomyelitis. The experiments of Teague and Goodpasture, Lipschütz and Flexner, suggest that the infectious agents of encephalitis, herpes simplex and herpes zoster are closely allied or are identical.

Infected foci of the upper respiratory tract are etiologic factors.

The description and distribution of the eruption in herpes zoster oticus is given. In typical cases, the geniculate ganglion of the seventh cranial nerve is involved, together with the motor branches, and often the eighth, ninth and tenth cranial nerves also.

The origin of all posterior spinal ganglions is the embryologic neural ridge; auditory ganglions also spring from the same source. The otic ganglion belongs to the sympathetic symptom and is not involved in herpes zoster.

Symptoms depend on the ganglions involved and are general and local. The auditory ganglions may escape or only one may be diseased. If Scarpa's ganglion is affected, the vestibular apparatus will be unexcitable. If Corti's ganglion is involved there may be only tinnitus or hypo-acusis or complete deafness may result. When there is pain and tenderness in the ear and mastoid with eruption on the drum membrane, otitis media is often suspected and paracentesis is done. One case is reported.

DR. JOHN R. PAGE, New York: We should be very careful to watch for these cases in the clinic. I am not qualified by my own experience to discuss this paper. None of the patients I have seen had facial paralysis. Patients with facial paralysis had no herpes. Dr. Kerrison speaks of the importance of making a prompt diagnosis in cases of involvement of the posterior part of the drum and posterior canal walls, in order to differentiate it from middle ear lesions. I would like to take the opportunity to congratulate Dr. Dennis on his fine demonstration of the subject.

DR. CROCKETT: I cannot remember ever having seen a case which approached the symptomatology of this one. I think Dr. Dennis should give us a report of this case a year later.

DR. RANDALL: One author reporting a case, mentioned that the picture was taken from the report of some other worker, as if he had not had any personal experience. He used the picture of a Boston drumhead as if New York did not have any normal drumhead. These cases are very rare. They are quite severe, and where ecchymoses occur it is a danger sign for vigorous careful treatment. Serious lesions about the eyes are likely to occur, but patients with ophthalmic zoster often have had malarial history and the condition often seems to yield to vigorous dosage of quinin. These conditions are frequently characterized by one diagnostic sign, that is, severe neuralgic pains for five or six days before the appearance of the herpes, which often gives rise to apprehension on the part of the patient. I have seen the occurrence of herpes in husband and wife, which developed on the sides nearest each other as they lay in bed, so in that case it appeared to be contact infection. This question has been raised by other workers. I believe in no previous case (unless possibly those which have been cited by Huntington) has there been anything like the careful analysis which Dr. Dennis has made in this case.

DR. JACK: I think this case is unique. I have no remembrance of any case with such a complexity of symptoms. It must be extremely rare.

DR. W. B. JOHNSON, Morristown, N. J.: I have seen cases of herpes but none of them with neurologic symptoms. I have never seen a case of herpetic irritation in the ear with this symptom complex.

DR. JOSEPH BECK, Chicago: Dr. Dench referred to my cases. In one, a man, aged 50, with peritonsillar abscess which developed while on a hunting expedition, the patient complained that his ear began to itch. I found a little black speck in the canal, with a slight red areola. The man went home; he then called me up to say that when he drank the fluid ran out of his mouth. I went to see him; by that time he had many eruptions and was feeling dizzy. I did not make the test for nystagmus. He had paralysis of the lower two branches of the facial nerve. His forehead could not be wrinkled. He went on to complete facial paralysis, he still has a paralysis and is deaf on that side. I removed the tonsils, hoping thereby to get some result but there was no change. I have seen other cases in consultation. In one case there was prompt result from the extraction of a diseased tooth; this was suspected of being the causative factor. There was an advanced corneal lesion in this case, but the recovery was perfect.

DR. ROBERT LEWIS, New York: I have seen two cases. I have one patient under observation, following extraction of a tooth, and presume the condition arose before the tooth was extracted. The herpetiform eruption was present when the patient was seen by me. There was involvement of the auricle, canal and drum membrane and entire half of the face. The gums and pharyngeal wall were also involved. There were no paralyses of the facial or other nerves. The deafness has gradually cleared up after three weeks. I saw another case two years ago. The patient came to me when the eruption on the face had disappeared. He had eruption of the canal and drum membrane with perforation of the drum membrane. This man had a facial paralysis but it almost entirely disappeared. These are the only two cases in my experience.

DR. T. W. WALKER, Fresno, Calif.: I have had a patient in whom the eruption extended up the forehead to the hair line. The right lower lid had ulcers on it and there was a very small corneal ulcer.

DR. FISHER: I remember Dr. Dench had a case similar to this which later turned out to be a cerebellopontile angle tumor. I do not think that a perverted horizontal nystagmus can be produced by anything but increased intracranial pressure. The sudden onset may possibly be accounted for by a localized arachnoiditis. I do not see how it could last so long, if the examination kept on showing the perversion of the nystagmus. I think if Dr. Dennis reports the case another year, a tumor of the angle will have been found. Duration of the symptoms does not mean very much. I have had one case of tumor; the growth has been there for eight years. The patient reported that he could not see well. He has choked disks. Tumors can lie quiescent for years without any development of importance. It is necessary to have a complete ear examination in cases of this type.

DR. DENNIS: I was unable to find a record of any case like this one with a complete vestibular examination. In such a condition, involving the neuritis of the eighth nerve, we do not know what the vestibular findings should be. I am impressed by Dr. Fisher's suggestion that it may be a case of angle tumor. I think we should have a complete report on these cases. With regard to treatment, apart from the suggestion of Dr. Randall, I have never seen treatment of any use. The eruption disappears, the pain disappears, and sometimes the paralysis disappears; sometimes it persists. In three advanced cases the paralysis persisted. One of the most significant things has been the suggestion that many cases of unexplained paralysis of the facial nerve and larynx may be abortive cases of zoster. Not all cases have the complete picture of herpes zoster oticus. It usually embraces the eighth nerve in the cochlear or vestibular branch, and sometimes there are seventh nerve symptoms.

THE ANATOMY, PSYCHOLOGY, DIAGNOSIS AND TREATMENT OF CONGENITAL MALFORMATION AND ABSENCE OF THE EAR. Presented by DR. BECK.

1. Introductory remarks concerning the sparseness of the subject in textbook as well as literature.

2. The psychologic examinations of these patients as well as the parents.

3. The psychiatric examination with special reference to the inferiority complex.

4. The medical or pediatrician's examination with special reference to the general physical examination and management.

5. The otologic examination with special reference to the external deformity, the radiologic examination and the physiologic examination, particularly as to the internal ear.

6. Lantern illustration of eleven cases on which this study is based.

7. The selection of five of these (*a*) infant, (*b*) older child, (*c*) adult, with their roentgenograms, functional tests and summary remarks.

8. Conclusions.

METHOD OF SKIN GRAFTING FOR RADICAL MASTOID CAVITY. Presented by DR. HARRIS P. MOSHER, Boston.

I prefer to graft a radical mastoid cavity whenever possible, and for the last ten years I have used the paraffin basket mold for the following reasons: it is an accurate mold of the cavity; it carries the graft to all parts, over the facial ridge, to the floor of the canal and fits the irregular walls of the middle ear; the mold can often be left in for several days, which is of especial advantage with children.

In cases in which secondary operation is preferable, the mold can be put in to control the granulations until the operator is ready to graft. It is also possible to graft only the middle ear, and do it accurately, with the use of the mold. Another advantage is that the paraffin adheres to the graft sufficiently to smooth out the wrinkles and prevent rolling of the edges. It has also been found that paraffin has the property of protecting the epithelium and stimulating its growth.

The question as to whether paraffin would impede drainage and so indirectly bring about meningitis or infection of the lateral sinus has been answered in the negative. The heat of the body melts the paraffin enough to permit drainage.

The method of making the paraffin gauze basket is as follows. Take a strip of gauze 6 inches wide, 12 inches long, fold twice toward the center, making a 4-ply tape 1½ inches wide; dip in melted paraffin so as to obliterate the mesh of the gauze. Roll up, place in a sterile container ready for use; when wanted unwind, softening if necessary in sterile warm water. Before using, the mastoid cavity is plugged with gauze soaked in epinephrin. A cone is formed of the paraffin tape, letting the cone edges overlap, the tip of the cone is filled with melted paraffin to take a mold of the middle ear and aditus. The gauze mesh must be thoroughly covered with paraffin or it will cause granulations. The mold is left in for hemostatic purposes while the graft is being cut. Then a second mold can be used to keep the cavity dry while the graft is being fitted on the first mold. The mold must be trimmed flush with the surface of the mastoid and not project, or it will interfere with primary healing. If a meatal flap is used it is tucked in place back of the mold, and its cavity is filled with ordinary gauze. The mastoid wound is closed, either wholly, or a portion can be left open for insertion of a drain. The mold is left in place for five to ten days. If the wound becomes puffy, red or discharging, the mold should be removed. To

remove, take out gauze packing and collapse the basket sides. When the mold is taken out it is a great satisfaction to see the well fitting graft left behind. The basket mold can easily be removed through the meatus.

In dressing the skin wound of the leg from which the graft was taken, a number of strips of paraffin gauze are cut and molded to form a plate. This is put over the skin wound and left in place for four or five days. The epithelium grows rapidly under the paraffin plate and is removed painlessly, thus forming a most satisfactory dressing.

(To be Continued)